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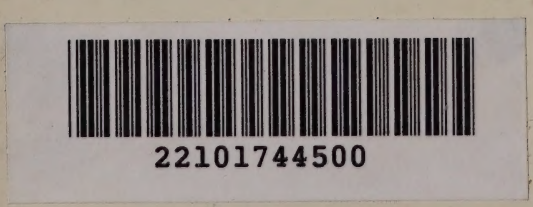

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CYCLOPÆDIA

OF THE

PRACTICE OF MEDICINE.

EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. V.

DISEASES OF THE RESPIRATORY ORGANS.

BY PROF. JUERGENSEN, of Tübingen; PROF. HERTZ, of Amsterdam;
PROF. RUEHLE, of Bonn, and PROF. RINDFLEISCH, of Würtzburg.

Translated by

GERALD F. YEO, M.D., of London; A. BRAYTON BALL, M.D., FRANCIS DELAFIELD, M.D., FRANK P. FOSTER, M.D., EDWARD FRANKEL, M.D., and JOHN C. JAY, JUN., M.D., of New York; and EDWARD W. SCHAUFFLER, M.D., of Kansas City.

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EDITOR OF ENGLISH TRANSLATION.

MANCHESTER MEDICAL SOCIETY

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BIOGRAPHICAL SKETCHES OF THE AUTHORS.

CARL SCHROEDER[1] was born on the 11th of September, 1838, in Neu-Strelitz (Mecklenburg), in which city he lived until he left the gymnasium. In the autumn of 1858 he went to Wurtzburg to pursue the study of medicine, and he remained in that city until the autumn of 1860, having completed four semesters; he then went to Rostock, where he passed his first examination in medicine. Returning in the summer of 1861 to Wurtzburg, he devoted three semesters to clinical study, and then in the autumn of 1862 for the second time returned to Rostock. A year later, in 1863, he passed his final examination, and in the winter received the degree of Doctor of Medicine. He immediately obtained the position of First Assistant at the Rostock Hospital, in the department of internal medicine, under the direction of Thierfelder. Meanwhile Veit, who had formerly been his preceptor at Rostock in the specialty that he was later to practise, having received a call to Bonn, in the place of Kilian, offered him the opportunity of taking the position of Assistant at the Obstetric Clinic in that city. Schroeder gladly accepted the offer, and remained there, a grateful pupil of Veit, from the spring of 1864 until the autumn of 1868, first as Special Assistant, and then, during the last semester, as Assistant at the Polyclinic. In the beginning of the semester of 1866 he obtained the position of Private Instructor in Obstetrics and Gynecology, his habilitation article being entitled: "Critical Researches on Retrouterine Hæmatocele," Bonn, 1866. A year later, he published in a separate book the results of his investigations and studies at the Lying-in Asylum of Bonn. The title was, "Pregnancy, Birth, and Childbed," Bonn, 1867. During this time, as well as later, he wrote quite a number of articles for the journals. In the autumn of 1868 he accepted the appointment of Extraordinary Professor at Erlangen, and Director of the Lying-in Asylum, in the place of Rosshirt, who resigned the directorship of the asylum in consequence of advanced years. In August of 1869 he was appointed Ordinary Professor. In the year 1870 his Treatise on Obstetrics appeared, of which a second edition followed in 1871, a third in 1872, and a fourth in 1874. He still holds the same appointment. Besides some minor publications, he published in the autumn of 1874, under the auspices of Ziemssen, a "Manual of Diseases of the Female Sexual Organs."

[1] This biographical sketch arrived too late for publication in volume X.—EDITOR'S NOTE.

THEODORE JUERGENSEN was born in the year 1840, in Flensberg, Schleswig-Holstein. In the year 1850 he entered the gymnasium of his native city, and having (in 1858) passed his maturity examination at the same institution, entered the University of Kiel. After residing there one year he went to Breslau, where, from 1859–61, he devoted himself exclusively to physiological and chemical studies under the supervision of Heidenhain and Lobhar Meyer. After remaining a semester in Tuebingen he returned to Kiel in 1862, undertaking the establishment and management of the Clinico-Chemical Laboratory. In the year 1863 he passed his state examination, received his degree, and established himself as a Private Instructor in clinical medicine. He was at the same time First Clinical Assistant at the Medical Clinic. As Instructor he devoted himself to all the branches of clinical medicine, but especially to physical diagnosis. In the year 1867 he became a practising physician, and as such had an extensive field of work. In 1869 Juergensen was appointed Extraordinary Professor, and assumed the supervision of the Polyclinic, which had been separated from the stationary clinic. In the year 1873 he accepted a call to Tuebingen, where he is now engaged as Ordinary Professor of Medicine, and Director of the Polyclinic. Some of his minor contributions are found in the "Deutsches Archiv für Klinische Medizin," in the "Berliner Klinische Wochenschrift," and "Deutsches Klinik." He has also written "Clinical Studies on the Treatment of Typhoid Fever by means of Cold Water," Leipsic, Vogel, 1866, and "Bodily Heat in Healthy Persons," Leipsic, Vogel, 1873.

Some of his lectures have also appeared in "Volkmann's Sammlung."

HUGO RUEHLE was born in 1824, in Liegnitz, in Silesia (Prussia). He studied in Berlin, under the special supervision of Traube; obtained his degree in May, 1846; then resided in Breslau, in the year 1851, as Second Physician of the All-Saints' Hospital, and from 1852 to 1857 as Assistant Physician at the clinic of Frerichs; then until 1860 as Physician in Chief of the same hospital. In the autumn of 1855 he established himself as an instructor at the University; in 1857 was appointed Extraordinary Professor, and in 1859 Ordinary Professor, lecturing on Physical Diagnosis, Pathological Anatomy, and Materia Medica. In the same year he assumed charge of the Polyclinic in Breslau. In 1860 he was sent to Greifswald as Niemeyer's successor. In the autumn of 1869 he was called to Bonn, in the same capacity as Director of the Medical Clinic, and this position he holds at present. His scientific works are as follows: In 1846 he published in "Traube's Beiträge," the first treatise on the mechanism of vomiting; in 1853 his habilitation essay appeared, the subject of which was the formation of bronchiectasiæ. In 1861 was issued "The Diseases of the Larynx," a clinical treatise. Other of his contributions on various subjects are to be found in the "Wurtzburger Zeitschrift," "Virchow's Archiv," "Greifswalder Med. Beiträge," "Volkmann's Sammlung Klinischer Vorträge."

GEORGE EDWARD RINDFLEISCH was born on the 15th day of December, 1836, in Koethen, Duchy of Anhalt. He is the third son of the Government Counsellor, G. Rindfleisch, and belongs to one of the old and distinguished families of the city of

Breslau. Daniel Rindfleisch, named Bucretius, was City Physician of Breslau at the time of the Thirty Years' War, and in this capacity drafted regulations for time of plague, which were even at that time highly valued. The services which he rendered anatomy were even greater, for he bought on his own account the seventy-eight plates of Julius Casserius, and, with Adrian Spigelius, published at Venice, in 1672, the book entitled: "De Corporis humani fabrica, libri decem." Rindfleisch, on his mother's side, is nearly related with the Von Meckel family of anatomists of Halle. His grandmother was the sister of Johann Friedrich von Meckel, whose services in the field of comparative and pathological anatomy caused him to be ranked among the best of his time. His grandfather, Wilhelm von Brunn, who brought up the lad, was body-surgeon to several successive princes and dukes of Anhalt, and while Superior Medical Counsellor of the duchy, drafted medical regulations which later served for the Prussian, and then recently for the German medical regulations. He studied several semesters in Heidelberg and Halle, then in the year 1856 followed the great magnet Virchow to Berlin, and under his guidance completed his studies. In 1861 he produced his public dissertation for installation as private instructor in Breslau, where, at that time, no chair of Pathological Anatomy had been established. In the spring of 1862 he was called to Zurich, to found a new chair in this department, being called from there in the autumn of 1865 to Bonn, where he was actively employed for eight and a half years. Since Easter of 1874, he has been engaged at the Institute of the University of Wurtzburg, the long celebrated nursery of medical science, and consecrated by the first achievements of his instructor Virchow.

Rindfleisch has written a large number of articles in Virchow's Archives, as well as in other medical periodicals, a monograph on "The Histology of the Blood," and a Treatise on Pathological Histology, which presents this matter for the first time in a systematic manner, and has therefore been adopted in all the high schools of Germany, and has been translated into many languages. Of the two English translations, one was published in Philadelphia (Lindsay & Blakiston).

ERRATA.

VOL. II.

On page 598, ninth line from the top, "intra-urine" should read "intra-uterine."
On page 607, seventh line from the bottom, "attacks" should read "attack."

VOL. X.

On page 360, top line, "between 50 and 70" should read "between 60 and 70."

CONTENTS.

Juergensen.

PAGE

CROUPOUS PNEUMONIA 4
History 4
Etiology 12
Pathology 32
Outline of the course of the disease 32
Outline of the morbid anatomy 40
Analysis of the individual symptoms 52
Chronology of the disease 52
Temperature 55
Symptoms of the circulation 61
Inspection 74
Mensuration 75
Palpation 75
Percussion 78
Auscultation 82
Condition of nutrition 92
Organs of digestion 94
Kidneys and urine 97
Abnormal substances in the urine 99
Condition of the skin.—Eruptions 100
Cerebral and nervous symptoms 101
Blood and hemorrhages.—Complications 106
Differences in the course of the disease 106
Forms of Pneumonia.—Relapses 106
Terminations.—Sequelæ 106
Diagnosis 134
Prognosis 138
Treatment 143

(Translated by A. Brayton Ball, M.D.)

CATARRHAL PNEUMONIA 183
History 183
Etiology 186
Pathology 190

PAGE

Symptomatology.—Course of the disease in general 190
Anatomical changes 194
Analysis of the symptoms 200
Complications and sequelæ 212
Diagnosis 216
Duration.—Terminations.—Prognosis 220
Treatment 223

(Translated by Edward Frankel, M.D.)

HYPOSTATIC PROCESSES IN THE LUNGS 236
Pathogenesis and Etiology 236
Pathological anatomy 240
Symptomatology and diagnosis 241
Treatment 243

PNEUMONIA FROM EMBOLISM 245
Pathogenesis and Etiology 245
Pathological anatomy 252
Symptomatology and diagnosis 254
Prognosis 258
Treatment 259

(Translated by Edward W. Schauffler, M.D.)

Hertz.

ANÆMIA 264

HYPERÆMIA AND ŒDEMA 265
Etiology 265
Pathological anatomy 270
Symptomatology 276
Course and termination 281
Prognosis 282
Diagnosis 283
Treatment 284

HEMORRHAGES OF THE LUNGS 289
History 290
Etiology 292
Pathological anatomy 298
Symptomatology 302
Duration, termination, and sequelæ 307
Prognosis 311
Diagnosis 312
Treatment 316

ATELECTASIS 322
History and literature 322

PAGE

Definition and etiology 324
Pathological anatomy 326
Symptoms and course 330
Prognosis 333
Diagnosis 334
Treatment 335

ATROPHY 337
Definition and etiology 337
Pathological anatomy 338
Symptomatology 340
Diagnosis 341
Treatment 342

HYPERTROPHY 342

(Translated by John C. Jay, Jr., M.D.)

PULMONARY EMPHYSEMA 344
History 345
Definition 346
Etiology 347
Theory of the development of emphysema 352
General features of the disease 360
Pathological anatomy 366
Relations with other diseases 375
Analysis of individual symptoms 376
Duration, results, and prognosis 391
Diagnosis 393
Treatment 396

(Translated by Frank P. Foster, M.D.)

GANGRENE OF THE LUNGS 407
History and literature 407
Etiology 408
Pathological anatomy 412
Symptomatology 415
Progress of the disease 420
Complications and termination 422
Prognosis 424
Diagnosis 426
Treatment 428

NEW-GROWTHS IN THE LUNGS 433
Pathological anatomy 433
Symptomatology 437
Analysis of the several symptoms 440
Prognosis 442

PAGE

Diagnosis 442
Treatment 443

NEW-GROWTHS IN THE MEDIASTINUM 444
Etiology 445
Pathological anatomy 446
Symptomatology 450
Analysis of the several symptoms 453
Duration, result and prognosis 456
Diagnosis 457
Treatment 460

PARASITES OF THE LUNGS 462
Animal parasites 462
Echinococcus 462
Strongylus longevaginatus, Diesing 467
Cysticercus cellulosæ 468
Vegetable parasites 468
Pneumonomycosis of Virchow 468

(Translated by Gerald F. Yeo, M.D.)

PULMONARY CONSUMPTION 473
Introduction 475
History 477
Etiology 481
Pathology 508
General description and course of the disease 508
Analysis of individual symptoms 517
The cough 517
The expectoration 520
Hæmoptysis 524
Pain 528
Dyspnœa 529
Inspection of the thorax 531
Other physical signs 532
Percussion 534
Auscultation 536
Laryngeal symptoms 543
Symptoms of the organs of circulation 548
The lymphatic system 550
The stomach 551
The intestinal canal 554
The liver 558
The spleen and kidneys 559
Nervous symptoms 560

PAGE
Cutaneous symptoms ... 561
The nails ... 563
The hair ... 564
The emaciation ... 565
The fever ... 567
Complications ... 572
Pneumothorax ... 576
Acute peritonitis ... 578
Gastric symptoms ... 579
Amyloid degenerations ... 580
Diagnosis ... 582
Course and termination ... 591
Prognosis ... 595
Treatment ... 600
Prophylaxis ... 601
Treatment of the chronic pneumonia ... 605
Treatment of the broncho-pneumonic form ... 607

ACUTE MILIARY TUBERCULOSIS ... 614
Introduction ... 614
Etiology ... 616
General description and course of the disease ... 618
Morbid anatomy ... 620
Analysis of individual symptoms ... 622
Complications ... 625
Diagnosis ... 626
Duration, terminations, prognosis, and treatment ... 629

(Translated by A. Brayton Ball, M.D.)

Rindfleisch.

CHRONIC AND ACUTE TUBERCULOSIS ... 633
Tuberculosis and scrofula ... 633
History ... 633
Scrofula ... 635
How scrofulous persons become tuberculous ... 639
Disease of the lymphatic glands ... 640
Anatomy of tubercles ... 642
Definition of tubercle ... 643
The origin of tubercle ... 644
Conclusion ... 646

CHRONIC TUBERCULOSIS.—PHTHISIS ... 648
Commencement of the disease ... 648
Catarrhal bronchitis and tuberculosis ... 648
The tubercle granule ... 648

PAGE

Erroneous explanations of the tubercle granule 653
Catarrh of the bronchi 653
The apices of the lungs 654
Hemorrhages and pleurisy 656
The initial hæmoptysis 656
Pleurisy 658
The later stages of the disease: Cheesy broncho-pneumonia 660
The crude tubercle of authors 660
Lesions of the mucous membrane 661
Tubercular ulceration of the mucous membrane 662
The peribronchial lesions 663
Desquamative pneumonia 664
Principal varieties of pulmonary phthisis 668
The ordinary chronic variety 668
The acute varieties 670
Atelectasis 670
Œdema 671
Slate-colored infiltration 672
Cheesy lobular masses 672
Cheesy lobar pneumonia 673
Cavities 676
Definition 676
Bronchiectatic conditions 676
The early stages of the formation of cavities 677
The opening and enlargement of cavities by the mechanism of respiration. 678
Enlargement of cavities 679
The fully-formed cavities 681
Healing processes 682
The rarer varieties 687

ACUTE TUBERCULOSIS 691

(Translated by Francis Delafield, M.D.)

CROUPOUS PNEUMONIA,

CATARRHAL PNEUMONIA,

HYPOSTATIC PROCESSES IN THE LUNGS,

AND

EMBOLIC PNEUMONIA.

JUERGENSEN.

CROUPOUS PNEUMONIA.

(This list of works includes only those which I have used.)

General: *Laënnec*, Traité de l'auscultation médiate, etc. IV. édition augmentée par Andral. Paris, 1837.—*Stokes*, On Diseases of the Chest. 1837.—*Grisolle*, Traité de la pneumonie. II. édition. Paris, Baillière et fils, 1864.—*Magnus von Huss*, Die Behandlung der Lungenentzündung und ihre statistischen Verhältnisse. Aus dem Schwedischen von Dr. J. Anger. Leipzig, Engelmann, 1861.—*Hugo Ziemssen*, Pleuritis und Pneumonie im Kindesalter. Berlin, Hirschwald, 1862.—*Lebert*, Klinik der Brustkrankheiten. Bd. I. Tübingen, Laupp, 1874.—*Wunderlich*, Handbuch der Pathologie und Therapie. III. Bd., 2. Abthlg. Stuttgart, Ebner und Seubert, 1856.—*Niemeyer*, Lehrbuch der spec. Pathologie und Therapie. Bd. I., 8. Auflage. Berlin, Hirschwald, 1871. Aerztlicher Bericht des k. k. allgemeinen Krankenhauses zu Wien. Jahrgg. 1858–70. Berichte der k. k. Krankenanstalt Rudolph-Stiftung in Wien. Jahrgg. 1866–69 und '71.—*Traube*. Gesammelte Beiträge zur Pathologie und Physiologie. Bd. II. Berlin, Hirschwald, 1871.

Historical: *Hippokrates*, ed. Kühn. Lipsiae, Cnobloch. 1825–27.—*Aretaeus*, übersetzt von Mann. Halle, Pfeffer, 1858.—*Celsus*, ed. Almeloveen. Basileae Thurneissen, 1748, ed. II.—*Caelius Aurelianus*, ed. Amman. Venetiis, Storti, 1757.—*Alexander Trallianus*, ed Guinther-Andernac. Basileae, Petrus, 1556.—*Rhazes*, Continens per clarrissimum doctorem Hieronymum Surianum. (Fol. sine loco et anno, Ende des 15. Jahrh. Tübinger Universitätsbibliothek.)—*Avicenna*, Canon e. c. per Fab. Paulinum Utinensem. Venetiis apud Juntin, 1608.—*Thomas Sydenham*, Opera. Medica. Translated by Dr. R. G. Latham. Sydenham Society Series.—*van Swieten*, Commentaria in Boerhaave aphorismos. Hildburghusae et Meiningae, 1747. Hanisch.—*Burserius*, Institutiones med. pract. Lipsiae, Fritzsch, 1790.—*Stoll*, Ratio medendi. Viennae, 1790, Kraus. —*J. P. Frank*, Grundsätze über die Behandlung der Krankheiten des Menschen. Mannheim, Schwan und Götz, 1794.—*Sprengel*, Handbuch der Pathologie. Leipzig, Schäfer, 1796.—*A. G. Richter*, Die specielle Therapie. Berlin, Nicolai, 1821. III. Auflage.—*S. G. Vogel*, Handbuch der practischen Arzneiwissenschaft. Wien, Lechner, Haykul, 1828. IV. Auflage.—*C. G. Neumann*, Von den Krankheiten des Menschen. Berlin, Herbig, 1832.—*Hufeland*, Enchiridion medicum. Berlin, Jonas, 1836.

Etiology: *Hirsch*, Handbuch der historisch-geographischen Pathologie. Erlangen,

Enke, 1859–64.—*Wilhelm Ziemssen*, Deutsche Klinik, 1857; Archiv für physiologische Heilkunde 1857; Prager Vierteljahrschrift 1858. (15. Jahrgang., 2. Bd.) These extensive and very carefully elaborated works present an excellent compilation of etiological material.—*Haller*, Denkschriften der k. k. Academie der Wissenschaften. Mathem.-naturw. Classe. Bd. 18. Wien, 1860. —*Oesterlen*, Medicin. Statistik. Tübingen, Laupp, 1865.—*J. E. Veith*, Handbuch der Veterinärkunde. Wien, Strauss, 1831. III. Auflage.

PATHOLOG. ANATOMY: *Rokitansky*, Handbuch der pathолog. Anatomie. Wien, Braumüller und Seidel, 1842–46.—*Rindfleisch*, Lehrbuch der pathолog. Gewebelehre. Leipzig, Engelmann, 1873. III. Auflage.—*Buhl*, Lungenentzündung, Tuberkulose und Schwindsucht. München, Oldenbourg, 1872.—*Cohnheim*, Neue Untersuchungen über die Entzündung. Berlin, Hirschwald, 1873.

PATHOLOGY: *Wunderlich*, Das Verhalten der Eigenwärme in Krankheiten. Leipzig, Wigand, 1870. II. Auflage.—*Wintrich*, Handbuch der speciellen Pathologie und Therapie, redigirt von Virchow. Bd. V., 1. Erlangen, Enke, 1854.—*Skoda*, Abhandlung über Percussion und Auscultation. Wien, Seidel und Sohn, 1864. VI. Auflage.—*Neubauer-Vogel*, Analyse des Harns. Wiesbaden, Kreidel, 1867. V. Auflage.—*Köhler*, Handbuch der speciellen Therapie. Tübingen, Laupp, 1867. III. Auflage.—*Jürgensen*, Grundsätze für die Behandlung der croupösen Pneumonie i. d. Sammlung klin. Vorträge, herausgegeben von R. Volkmann. Leipzig, Breitkopf und Härtel, 1872.—Articles in journals and works of minor importance are referred to in the text.

HISTORY.

In its fully developed form croupous pneumonia belongs to those diseases which, on account of the constant recurrence of certain sharply defined characteristics, are recognized even by the non-professional public as special and independent affections. The frequent occurrence of inflammation of the lungs affords abundant opportunity for observing the disease. The people are therefore familiar with it, and call it by names of their own, which are usually borrowed from scientific language, and altered into more easily pronounceable forms. In northern Germany, for instance, "Fleier" means especially inflammation of the lungs, but includes also every acute affection, in which pain in the side is present. "Fleier" is a corruption of pleuritis. The popular view has therefore been the same as that which has prevailed among medical men from the most ancient times up to the period when the science of physical diagnosis became generally known. The doctrine of pneumonia presents three stages of development.

In the first stage the attempt was made to separate pleuritis (in its old signification) from pneumonia, but the desire was greater than the ability. In the second stage, this difficulty was recognized; in consequence of the defects in the methods of diagnosis, the distinction between the two diseases had hitherto been merely a question of amount. Laënnec was the first who made it possible to draw the lines between them. The advance of science narrowed the limits of the true croupous pneumonia more and more. We stand at the beginning, not at the end, of the third stage, during which it is to be hoped that the superstructure of our knowledge will rest upon the foundation of the methods of physical science. Until recently croupous pneumonia belonged to those diseases which were supposed to be accurately understood, and yet more thorough study has gradually revealed the imperfection of our knowledge. Of this fact my present task will afford more proof than is agreeable to me. I give below a brief historical sketch.

Among the Greek and Roman writers "Pleuritis" and "Peripneumonia" comprised the sum of their knowledge of this class of diseases. They were regarded as distinct from each other, but we should err if we supposed that these terms correspond to ours: "Peripneumonia" to our croupous pneumonia, "Pleuritis" to our inflammation of the pleura. On the contrary, there is a continual confusion between the two conditions; other affections are often classed with them, which the anatomy and physiology of our day oblige us to regard as distinct. We shall not be far from the truth if we regard peripneumonia as comprising the more severe affections of the thoracic viscera, and pleuritis the less severe, and the latter only when accompanied by pain in the side.

I quote a few authorities upon this point:

Hippocrates.—Allusions to pulmonary affections are found scattered through many parts of the so-called writings of Hippocrates. In addition to some admirable remarks, which display a fine talent for observation, we come across such odd conceits as the diagnosis of the seat of the pneumonia by the coating of the tongue, and the belief that individuals accustomed to hard labor succumb to the disease more readily than those in delicate health (Coan Prognostics).

From his description of peripneumonia in the Prognostics, it would be difficult to diagnosticate a pneumonia as we now understand it.

Aretæus.—In the two chapters on "Acute Diseases," pleuritis and peripneumonia are represented as distinct affections. The former is described as an inflammation of "the thin, but firmly adherent membrane called the girdle (die umgürtende), which lies underneath the ribs and spinal column, within the cavity of the chest, and extends as high as the clavicle." The symptoms are stated to be high fever, sharp pains shooting as high as the clavicle, dyspnœa, sleeplessness, anorexia, bright flush on the cheeks, dry cough, viscid sputa, and expectoration of yellow, bilious, or bloody mucus. The fact is also mentioned that the patient lies upon the diseased, and not on the sound side, on account of the dragging produced by the weight of the inflamed membrane. Among the *symptoms of peripneumonia* we find mentioned: "High fever and oppression in the chest, but no pain, if the lung alone be affected; but if the inflammation extend also to the enveloping membranes the pain is never absent. The respiration is very difficult, and the expired air hot. Such patients prefer to sit upright in bed, because the position is the most comfortable for breathing. The face is red, especially the cheeks; the white of the eye has a greasy lustre. The tip of the nose is turned upwards. The veins of the temples and neck are prominent. The appetite is poor. The pulse is at first large, empty, very frequent, and bounding. The external parts are moderately warm and moist, the internal warm and dry. The spirits are depressed. The cough is generally unaccompanied by expectoration, but when sputa are discharged they consist of a mucus, which may be frothy, or strongly bilious, or *tinged with bright red blood.* This blood-red mucus is the worst of all the signs." If the disease tend to a fatal termination, the symptoms are sleeplessness, coma, moderate delirium, cold extremities, blueness of the nails, and a small, very rapid, intermitting pulse. Most of the cases die on the seventh day.

Pleurisy attacks most frequently the aged, less frequently young and middle-aged persons, rarely children. The mortality of the aged is small; they regain their health very readily; so also do children, but young and middle-aged persons escape more rarely. Cases of recovery from peripneumonia are as uncommon among young persons and robust adults as from phthisis among the aged.

These extracts, notwithstanding the admirable observations on the individual symptoms, show how confused a conception of pneumonia was entertained by one of the ablest physicians of antiquity. It is surprising that he should have given a favorable prognosis for the aged, and an unfavorable one for vigorous persons. The remarks of Celsus upon this subject are still more unsatisfactory.[1] The description given by Cælius Aurelianus is much more comprehensive; he also treats of pleuritis and peri-

[1] Lib. IV., Cap. 6, 7.

pneumonia in separate chapters. As his account is too long for quotation *in extenso*, I present merely an abstract.

Pleuritis: Burning fever, severe pain in the side, which may extend to the clavicle and scapula of the affected side, even to the arm, chest, or groin, and exhibits many changes in the forms of its manifestations. Slight cough and dyspnœa are also noticed; the expectoration may be absent, but is usually present, at first frothy, afterwards bloody or bilious, finally purulent. Decubitus on the healthy side is uncomfortable, on the diseased side, painful. The patient is sleepless; his tongue dry and rough.

Peripneumonia: Burning fever, a feeling of oppression in the lateral and middle portions of the chest, a tendency to lie with the body partially erect and bent backwards, relief on sitting upright, a sense of suffocation upon lying on the side. The color of the face is red, almost florid; the expression of the eye languid, unsteady, and yet possessing a certain brilliancy. Respiration frequent, slight cough, with bloody or bilious expectoration. The sputa, which are frequently soot-colored, are more yellow and frothy than those of pleurisy. The respiration is difficult, and the patient feels the need of breathing hurriedly, he inhales as much as possible, and prefers air which is cold. The mouth is dry, the tongue rough, at first whitish, afterwards red. The pulse is strong and rapid. Anxiety, restlessness, constant insomnia. If sleep occur it is uneasy, and disturbed by groans. In brief, *the chief symptoms of peripneumonia are*, high fever, difficult and rapid breathing, cough, attended by various kinds of expectoration, a feeling of oppression in the chest, without any, or with only slight pain. Dread of suffocation.[1]

Alexander Trallianus, who was also a prominent writer, gives the following description:

Pleuritis: High fever, dyspnœa, stabbing pain, cough; these symptoms point positively to a pleuritis.[2]

Peripneumonia is not called by this name; in place of it the more general term, "inflammation of the lungs," is preferred. Its symptoms are: dyspnœa, rapidly ensuing burning fever, sometimes also a coated tongue, flushed cheeks, and a sense of oppression (in the breast). In general, even if the expectoration be very scanty, and the breathing difficult, all these symptoms necessarily indicate the occurrence of an inflammation in the lungs.[3]

These brief quotations are sufficient for my purpose. It has seemed to me very strange that the Greek and Roman physicians possessed no clear knowledge of the typical course of pneumonia; and this all the more because they were not deprived of

[1] Acutor. morbor. Lib. II., Cap. 13–29.

[2] Lib. VI., Cap. 1.

[3] Lib. V., Cap. 2.

such information by their mode of treatment, which was far less meddlesome than that of later times. Blood-letting, especially, was practised to only a very limited extent.

As a matter of course, but little progress is to be looked for among the Arabians, as is shown by the writings of their principal authors, Rhazes and Avicenna.

In Rhazes the passage on this subject is found in the Continens.[1] He quotes Aaron, and contents himself with adding some nice discriminations in regard to the pulse. Avicenna,[2] it is to be noticed, holds that peripneumonia and phthisis may be caused by pleuritis, and, on the other hand, pleuritis by peripneumonia. The chief importance is ascribed to the nature of the sputa, in regard to which a great number of refinements are made, which have no foundation in fact; but the main point, the bloody character of the expectoration in pneumonia, is overlooked. They who wish to find an allusion to the crisis may find it in his very brief statement, "and thus it (peripneumonia) kills up to the seventh day," quoted, probably, from Aretæus, who makes the same observation.

Let us now, with a long stride, pass to a more recent period, as the intervening centuries are unprofitable for study. If any one wishes to acquaint himself with the opinions of those times, he will find abundant material in the Brissot controversy on venesection. In van Swieten's commentaries on Boerhaave, considerable space is devoted to the consideration of pleuritis and peripneumonia, but no distinction is made between them.

Moreover, even the inflammation of the internal intercostal muscles is defined as "pleuritis vera" (§ 878). This is entirely in accordance with the definition of pleurisy, which regards the pain as the chief element, and confines itself merely to symptoms. On the other hand, the definition given of pleuropneumonia is a causal one; "a true inflammation originating in the pulmonary vessels, which are particularly disposed to inflammation" (§ 820). So strong were these preconceptions that even palpable facts could not remove them. In thirty autopsies on persons who were supposed to have died of pleuritis, Petrus Servius found only one lung diseased in every case, and the pleura unaffected. The explanation given was that the primary disease was the pleuritis, and that the lung had been affected secondarily in consequence of the imperfect respiration (§ 877). In other respects the chapters devoted to this subject are full of details of the highest interest. That mistakes very often occurred, may be inferred both directly and indirectly. Boerhaave himself mentions one case of empyema, which he mistook for peripneumo-

[1] Lib. X., Cap. 1.

[2] Canon, Lib. III., Tractat. IV., Cap. 1.

nia; the autopsy cleared up the case. On another occasion a mistake was caused by bronchiectasis (§ 836).

For every physician who wishes to gain an insight into the state of medical science among the eminent men of antiquity, before the revival of knowledge in our times upon the basis of the physical sciences, these commentaries possess the greatest interest.

Although an apparently retrograde step, it was in reality a positive advance when scholastic definitions were discarded, and the identity of pleuritis and pleuropneumonia was asserted. So far as I am familiar with the literature of those times, I think the credit for this is to be given especially to Borsieri. His expositions of the subject are at any rate free from ambiguity, and reached a wide circle of readers by means of his text-book.[1]

Sydenham, it is true, had already hinted at the relation between the two diseases, but he does not seem to have examined the question very thoroughly. The passage reads: "Inflammation of the lungs, which, in my opinion, differs from pleurisy only in its greater intensity, its more violent cause, and wider prevalence" (l. c., volume first). In Borsieri the following explicit passages are found: "*Inflammation of the lungs is of two varieties, peripneumonia and pleurisy; these affections, in my opinion, do not differ in their situation or nature, but only in the character of their symptoms*" (l. c., Vol. IV., § 97); and in another place: "*The only distinction between peripneumonia and pleuritis, if there be any, is the violent and continuous pain*" (§ 100).

These quotations show very clearly what a change of opinion had taken place in regard to the distinction between the two diseases, and it will now be very interesting to notice the efforts to explain and to subordinate to the fundamental conception the difficulties which really existed, such as the fibrinous deposits upon the lungs, and exudations into the pleural cavity, which were found in persons who had died of peripneumonia.

Borsieri held, with many of his contemporaries, that the first changes in peripneumonia were to be sought for in the blood, which he supposed was partially converted into a "gelatinous mucus" (mucosum gluten). In describing the termination of the disease he says: "As a consequence of such contraction of the chest and closure of its (natural) passages, the mucous fluid everywhere exudes in large quantities through the exhalant vessels, and cannot be taken up again by the

[1] Institutiones medicin. practic.

absorbents, because the latter are over-distended. Thus it happens that the mucous fluid collects about the lungs, mediastinum, pleura, and pericardium, coagulates in the cooling body after death, and at the autopsy appears as a more or less extensive layer of mucus overlying these parts. Again, if the blood be retarded, or altogether stopped in its circulation, its serous part is separated, and exudes into the cavity of the chest, and then a *hydrothorax* is superadded to the pneumonia. This condition is called *hydropneumonia*" (§ 106).

In the histories of cases and of the results of their autopsies which Stoll[1] has published, we have a good picture of the diagnostic skill at that time in recognizing pneumonia in the living subject. In cases which were not characteristic this task was by no means an easy one, even for the most skilful.

I quote only a few examples:—Bedside diagnosis: Febris biliosa cum inflammatione thoracis complicata. Autopsy diagnosis: Pneumonia lobuli inferioris later. dextr. (Histor. morbi, VI.). Bedside diagnosis: Febris maligna. Autopsy diagnosis: Pneumonia lob. superior. dextr. (Historia morbi, XXII.).

The view of Borsieri, which was shared by Tissot, Sarcone, and other prominent contemporaries, was for a long time generally accepted. In the manuals and text-books which appeared up to 1820, their ideas are expressed more or less distinctly. I shall here refer to only a few of them.

"*If an inflammatory fever be accompanied by intense stabbing or oppressive fixed pains in the chest, and by great dyspnœa, severe cough and expectoration, we call the disease inflammation of the chest, or an inflammation of the lungs, without admitting the distinction, which has been commonly made since the time of Diocles, between pleurisy and peripneumonia.*"[2] "*It seems clear, therefore, and indisputable, that there is no distinction between these diseases (pleuritis and pneumonia), and that we can and ought to discuss both of them under the single term peripneumonia.*"[3] "*Inflammation of the lungs and acute pleurisy (peripneumonia, pleuritis, and pleuroperipneumonia). Neither in their essential nature nor in their symptomatology can these two inflammations ever be accurately differentiated from each other. The attacks of pleuritis are always associated more or less with peripneumonia, and, on the other hand, the symptoms of the latter with those of the former. In many cases we cannot say which has the upper hand; and at the autopsy we usually find all parts of the cavity of the chest inflamed. The two inflammations ought not, therefore, to be sep-*

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[1] Ratio medendi, Pars VII., Sect. II.

[2] *Kurt Sprengel*, l. c., p. 347.

[3] *J. P. Frank*, l. c., Bd. II., p. 118.

arated from each other, and what is said of one will, with few exceptions, answer for the other." [1]

With Laënnec begins a new era in the theory of inflammation of the lungs. His anatomical and clinical descriptions are clear and true to nature. Here is the turning-point, as must be evident to every one who has tried to enter into the views of that period, which, with all its talent, still lacked the one thing needful, the scientific method. In Laënnec's descriptions there may be here and there a statement that is not quite satisfactory; yet upon the whole it may be said that he laid the scientific foundation for future investigation in the theory of chest diseases in general, and particularly of pneumonia. In the construction of this theory France and England took a temporary precedence of Germany, where a long time elapsed before these views were generally received and acted upon.

They were opposed by the old men. Thus Hufeland, in his Enchiridion Medicum, the preface of which is dated May, 1836, uses the following expression: "In more recent times, the signs derived from hearing with the aid of the stethoscope or percussion, have been much recommended for the diagnosis of chest diseases. These signs are, however, very fallible, and by them alone we can never discover the existence of an inflammation, unless we avail ourselves of those other symptoms, which are of themselves sufficient for a diagnosis." The diagnostic criteria which Hufeland gives could just as well have been written several centuries before.

The Vienna school, especially Skoda, were the first to naturalize in Germany the physical methods of diagnosis. The Viennese are to be credited, however, with something more than the mere promotion of diagnostics; we owe to them the doctrine of pneumonia. Rokitansky has elaborated in a final manner the macroscopic part of the pathological anatomy. The skepticism of the Viennese in therapeutic matters has, however, been unable to overthrow the treatment of pneumonia by venesection, a practice confirmed by the authority of many centuries (Dietl).[2]

By all these changes of opinion the church-like quiet which, since the time of Hippocrates, had almost continuously settled

[1] *A. G. Richter*, l. c., Bd. 4, pp. 372-3; also *S. G. Vogel*, l. c., Bd. 4, p. 146 et seq. *Neumann*, l. c., Bd. I., p. 151 et seq.

[2] Der Aderlass in der Lungenentzündung. Wien, 1849.

about the subjects of pleuritis and peripneumonia, has been dispelled. Modern science now tests its strength against one of the primevals, one of the mighty men of a past, which supposed that just here the ground under its feet was most secure.

ETIOLOGY.

Croupous pneumonia belongs to those diseases which are very widely distributed. On an average, 3 per cent. of all diseases in the whole population of the globe are due to this cause. In comparison with internal diseases alone, it comprises in Germany, England, and France about 6.4 per cent.

Hospital statistics show that, as a rule, more than 2 per cent. of the total hospital admissions are cases of pneumonia. In Vienna the proportion is 2.6 per cent., in Berlin 2.5 per cent., in Stuttgart 2 per cent. But since children and old persons are in general not received by the larger institutions, these numbers do not express the whole truth. The polyclinics and the practice of the physicians to the poor are more reliable sources of information. According to the compilations of Ziemssen, these sources report about one per cent. more cases than the hospital records, *i.e.*, somewhat more than 3 per cent. At the polyclinic in Kiel my cases of pneumonia amounted to as much as 5 per cent. of the whole number of patients—203 out of 3,993.

Pneumonia ranks high also among the causes of death; about 6.6 per cent. of the total mortality is due to this disease, about 12.7 per cent. of the mortality from internal diseases. The so-called secondary pneumonias are not included in these percentages.

The significance of these figures, which were compiled from about half a million cases of fatal pneumonia, is best seen by a comparison with other diseases.

For every 100,000 inhabitants there die yearly –

	London.	Paris.	Berlin.
From phthisis	323	382	296
" pneumonia	170	252	113
" bronchitis	115	172	38
" typhus	95	103	90

Pneumonia is thus seen to rank next to phthisis as one of the most dangerous enemies to human life.

Pneumonia occurs in all degrees of latitude, and does not seem to be absolutely more frequent in one latitude or climate

than in another. The widest differences in mortality are often shown by places which are very near each other, and possess the same climatic conditions; and, on the other hand, the same ratios are found in localities which are widely separated, and have an entirely dissimilar climate.

In proof of these statements I shall give only a few of the numerous facts which Ziemssen, with whom Hirsch in the main agrees, has collected. In Ireland the mortality from pneumonia is 0.3 per thousand inhabitants, in England 1.3. In the West Indies, among the English troops, it varies between 0.3 (Jamaica) and 3.7 (in the Bahamas). Hamburgh and Turin exhibit the same mortality, likewise New York and Copenhagen.

The geographic distribution of pneumonia is entirely different from that of catarrh and bronchitis. The latter affections increase in frequency the farther we advance from the tropics to higher latitudes; this is not the case with pneumonia. Nor, as has been supposed by many, is the reverse of this statement true.

Upon this point Ziemssen and Hirsch are in entire agreement; in fact the statistics we already possess are amply sufficient to establish it. Its importance need hardly be discussed here.

The season of the year has an undoubted influence upon the prevalence of pneumonia. In considering the season-rates in Europe, we must distinguish between *the climate of the coast* and *that of the continent*, because in each of them a special mode of distribution is to be noticed. The general fact, however, is common to both, that about two-thirds of the cases of pneumonia occur in the *winter* and *spring months*, and about one-third in those of *summer* and *autumn*.

	Winter.	Spring.	Summer.	Autumn.
Edinburgh.....	37.6 p. ct.	26.1 p. ct.	16.7 p. ct.	19.6 p. ct.
Dublin.....	36.0 "	28.1 "	15.9 "	20.1 "
Copenhagen.....	29.9 "	33.0 "	19.6 "	17.6 "
Berlin.....	29.2 "	33.1 "	19.0 "	18.7 "
Zürich	32.9 "	41.9 "	12.4 "	12.8 "
Turin.....	33.0 "	36.5 "	13.2 "	17.3 "
Average	31.1 "	33.1 "	16.1 "	17.7 "
	66.2 p. ct.		33.8 p. ct.	

This table is constructed from the mortality reports of the respective cities for a long series of years. In a disease of such short duration as pneumonia, the number of deaths during these periods will give us an approximate idea of the number of patients ill with the disease. This fact is shown also by proper control calculations. Moreover, the difference between the different seasons becomes much more marked when larger numbers are at our command, and the year is divided into only two divisions. In the Vienna General Hospital, during a period of twenty-four years, there were received 12,104 cases of pneumonia: 64 per cent. in winter and spring; 36 per cent. in summer and autumn. In the Seraphim-Lazareth,[1] in Stockholm, 2,616 cases of pneumonia were admitted during sixteen years: the relative proportions were 61.5 per cent. and 39.5 per cent.

From these data the differences between an insular and a continental climate are very apparent. In Edinburgh and Dublin the maximum frequency occurs in winter, in the other places in the spring. The general fact is shown still more strikingly by the following comparison, which is also taken from the lists of mortality for the entire populations:

	Winter.	Spring.	Summer.	Autumn.
Insular climate: per cent. (England—Ireland).	36.0	25.9	15.7	22.4
Continental climate (Germany).	27.3	36.2	18.8	17.7

These figures show that in a *continental* climate the *maximum* frequency occurs from *March* to *May*, the *minimum* from *September* to *November;* in an *insular* climate, on the contrary, the *maximum* occurs from *December* to *February*, the *minimum* from *July* to *August.*

The statistics of morbility,[2] taken from hospital and other sources, also give essentially the same result. They scarcely, however, warrant any wider conclusions. It is to be noticed that the disease as seen on the coasts of the Baltic Sea conforms entirely to the continental type.

According to the tables of mortality, the *maximum* number of deaths for an *insular* climate occurs in *December;* for a *continental* climate, in *March* or *May.* The *absolute minimum* occurs most frequently in *September*, next in *August.*

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[1] *Magnus v. Huss*, l. c., p. 67.

[2] The German "Morbilität" is translated by the corresponding word morbility, in the sense of the number of individuals ill with the disease, as distinguished from the mortality, or number of those dying from it.—TRANSLATOR'S NOTE.

These authenticated figures do not, it seems to me, warrant the farther conclusions which Ziemssen has drawn from them. His statement that the maximum frequency of pneumonia shifts in point of time from west to east, and the minimum in the reverse direction, is still very problematical.

The rates in non-European countries have not been studied with enough care to give us a sufficiently large number of facts, but with our present knowledge they agree very well with the European statistics.

In the United States, of all the cases of pleuritis and pneumonia occurring among the regiments, at all stations, between 1839 and 1855, there were from January to June 18, 60 per cent.; July to December 12, 40 per cent.

For Europe the figures are: from January to June 18, 60.6 per cent.; from July to December 12, 39.4 per cent.

The same differences in type presented by the continental and insular climates in Europe are found to exist also in the tropics.

Pneumonia is affected in its frequency by the season of the year very differently from pleuritis and bronchitis.

This is shown by the mortality tables of London, from 1840 to 1854, which include 56,285 cases of pneumonia, and 2,090 of pleuritis.

	Jan.–March.	April–June.	July–Sept.	Oct.–Dec.
Pneumonia..	31.3 per cent.	21.4 per cent.	14.4 per cent.	32.9 per cent.
Pleuritis	28.9 "	26.8 "	18.2 "	26.1 "

Moreover, pleuritis is much more uniformly distributed throughout the year than pneumonia, and the maximum frequency of the two diseases does not occur at the same time.

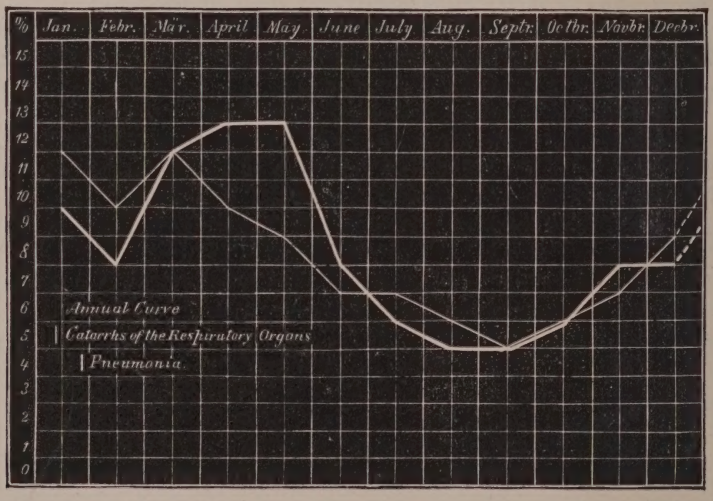
The ratio of catarrhs to pneumonias is shown by the following account of the admissions for twenty years to the General Hospital in Vienna, embracing 23,503 cases of catarrh, and 11,913 of pneumonia.

	Jan.–March.	April–June.	July–Sept.	Oct.–Dec.
Pneumonia.....	29.5 p. ct.	33.5 p. ct.	15.4 p. ct.	21.6 p. ct.
Catarrh of the respiratory organs...	33.8 "	26.7 "	17.5 "	21.9 "

These percentages are upon the whole nearly the same, but their non-coincidence becomes still more apparent by reference to the following annual curve drawn for Vienna, with the separate months indicated. (See next page.)

Whether the more severe climate of a locality, which is elevated and exposed to the winds, favors the development of pulmonary inflammations, is a question yet unsettled.

Hirsch and Ziemssen entertain the most antagonistic opinions upon this point. Hirsch, without giving us the figures, and referring partly to the accounts of travellers, states that pneumonia and pleuritis are of "frequent" occurrence in the American Andes, upon the plateaus of Arabia, and in the mountainous districts of Abyssinia, etc. That the rarefaction of the air does not, however, of itself favor the occurrence of pneumonia, is shown by the instance of the city of Mexico, which is situated 7,459 feet above the level of the sea. On the other hand, Ziemssen mentions Iceland, where the climate is certainly very severe, and yet the mortality of pneumonia is only thirteen per 1,000 deaths from all causes, that of pleurisy sixteen. In inclement and mountainous Wales there are only eight deaths from pneumonia for every 10,000 inhabitants; the mountainous counties in the north of England show only twelve, while in the level country of the adjacent southern counties the mortality is respectively sixteen and thirteen. Few cities in the world have so small a number of deaths from pneumonia as Munich, which is situated on high ground, and is exposed to the winds on all sides. Its mortality from pneumonia is only five deaths per 10,000 inhabitants. Ziemssen's data appear to me to be more numerous, more accurate, and therefore more trustworthy than those of Hirsch.



At all events it has not been demonstrated that an elevated position or the prevalence of stormy winds exerts any important influence.

The question very properly arises here, whether the *pressure* and *temperature* of the *air* play an important part in the etiology of pneumonia?

Probably the *height of the barometer* has some influence

upon the frequency of pneumonia. At least in some places the disease is less frequent when the barometric ranges are high. Whether this is a mere coincidence, or whether the connection between the two conditions is causal, remains to be determined.

Those writers who have more numerous barometric registrations at their command, express themselves very cautiously upon this point; for example Haller.[1] It has seemed to us in Kiel that pneumonia occurred more frequently when a continuous high range of the barometer, which is the ordinary condition there in the north-east and east, is suddenly interrupted by a considerable fall; but I have no records in proof of this opinion.

Neither an absolutely high nor an absolutely low *temperature* appears to exert much influence. Rapid changes, however, seem to produce important results.

In countries lying far to the north pneumonia is not especially frequent during their cold season, nor are the severe winters usually characterized by an increased mortality. The statistics already given for Iceland may serve as proof of the first part of this statement. Jos. Frank, from observations made by him at Wilna, states that pneumonia was not prevalent among the troops of Napoleon's army during the retreat in the severe winter of 1812, notwithstanding their exposure to all the inclemencies of the weather. Huss makes a similar statement for Sweden.

The distribution of the number of cases of pneumonia through the single months of the year, and the comparison of single years with each other in respect to the variations of the warmth of the air, lead to the conclusion that *rapid changes* in the *daily temperature* undoubtedly favor the production of the disease.

Huss has admirably succeeded in deducing this fact from the mass of his observations. Numerous observations have been made in other quarters with a similar result. I refer again especially to Haller.

The influence of the *moisture* of the *air* has not as yet been ascertained.

The facts upon this point are too scanty for a secure basis. "If we are willing to draw general conclusions from a small number of facts, then it may be inferred from the rare occurrence of inflammation of the lungs in some districts where the atmosphere is relatively very moist, that a high degree of moisture, in contrast with its positive influence in the production of catarrh and bronchitis, is anything but favorable to the prevalence of pneumonia" (Hirsch).

[1] Wiener Denkschriften, l. c., p. 9.

A *marshy malarial soil* does not appear to have any constant effect upon the frequency of pneumonia.

Grisolle has devoted special attention to this point, and has ascertained that in malarial districts the number of cases varies from time to time without any definite rule being noticed.

The difficulty of obtaining reliable data, great as it has been in the part of etiology previously discussed, increases enormously when we come to consider the individual ratios, the position of the individual in relation to the community. In this case we can hardly expect any other than a negative result, unless we adopt the naïve practice of compiling statistics which are accurate to a per cent., but include only a small number of cases occurring within a very circumscribed district (Kirchthurmhorizont), and then without further ceremony generalizing from them. Hospital reports, in other respects so valuable, furnish figures which hardly admit of comparison with each other, because the small fraction of the population which is found in these institutions is very variable in its social position in different places. Prejudices, customs, the greater or less variety of industrial occupations, these are incommensurable quantities which disturb and confuse when we wish to estimate their value. *We need mortality statistics which shall include all classes of society, and these can be obtained only from physicians who are in private practice.* Such statistics could certainly disclose to us facts which we little suspect, and which would be of great service to science. So long as we are without them our knowledge must be fragmentary.

Certain general conditions seem to be satisfactorily proved.

Those communities which lead mostly an out-door life suffer less from pneumonia than those which carry on their occupation in confined apartments.

Although this proposition is opposed to the generally received opinion, the evidence in favor of it is conclusive.

The average mortality from pneumonia for every 1,000 inhabitants is:

For the agricultural population of England in seven counties, exclusive of the cities, 0.8.

In the twenty-five largest towns in England, London not included, 2.0.

The average for the city and manufacturing population of England is 1.6.
For the population living out of doors in the same country, 1.0.
The same is true of other countries.

Country Population.			City Population.		
State of Maryland...	0.8 per 1,000.		City of Baltimore...	0.9 per 1,000.	
Province of Leinster.	0.3	"	" Dublin.....	0.8	"
Canton of Geneva...	1.0	"	" Geneva.....	1.1	"

These figures, taken from Ziemssen's work, are confirmed by Grisolle; at least they agree with the data published by him, to which, however, he does not himself attribute much importance.

Some classes, which are particularly exposed to the inclemencies of the weather, suffer but little from pneumonia. Grisolle states that the official lists show only 175 cases of pneumonia out of 24,000 sailors in the French marine, who were at sea in the most different latitudes, and of these cases the majority occurred not upon the open sea but near the coast. The English official reports corroborate this statement. Soldiers are attacked with pneumonia more frequently in the garrison than in the field, as is shown by the reports of the English, Russian, and French troops during the campaign in the severe climate of the Crimea.

On the other hand some classes, which are more or less withdrawn from the influence of the weather, are very frequently attacked by pneumonia.

The occupants of prisons suffer severely. According to the census of Ireland, in 1851, the number of cases of pneumonia in the hospitals was 1¼ per cent.; in the prisons, 2 per cent. The same observation has been made by many other writers. Moreover, the mortality from this cause in cloisters appears to be very high; in Paris the deaths among the nuns amounted to 17.02 per 1,000 inhabitants, while among the washerwomen it was only 3.05.

The disposition to this disease shown by individual occupations has not been definitely ascertained. The opinion that those who are exposed to the inclemencies of the weather are especially liable to attack is not proven.

It is commonly believed that laborers, whose occupations oblige them to be out of doors in all sorts of weather, furnish a large contingent of the cases; but this is quite positively disproved by the statistics already quoted in regard to the whole

population of England. If we at all carefully examine the details which writers give us on this point, we shall scarcely be inclined to form any other opinion. In the statistical tables of hospitals we have to consider a class of causes which cannot be represented by mere figures and percentages.

The remarks of Grisolle[1] upon this subject are well worth perusal. Of 1,264 cases of pneumonia admitted into the Paris hospitals during 1861, 482 were servants. But, says Grisolle, before we conclude from this fact that there is a special and strongly developed disposition to the disease in this class, we should inquire into the vital conditions under which they live. In Paris servants generally occupy such small rooms that when they become seriously ill it is impossible for them to be treated at home, and they are therefore obliged to seek public assistance and to go to the hospital. The same thing is true of masons, who pass only part of their time in the city and live without their families in confined hired rooms. With other occupations—cooks, conductors, draymen, porters, etc., it is different; they are less frequently received into hospitals, because they often belong to societies for mutual support or for distribution of poor funds, so that in case of sickness they are able to remain at home.

Grisolle also shows us how often a tradition in such matters may lead us into error. Among 670 laborers engaged in unloading vessels, and frequently exposed to cold and wet, Parent-Duchatelet, after careful examination, found only one man who had had a "fluxion de poitrine." Grisolle himself found among sixty-eight women, thirty-three who worked in close rooms and thirty-five who had out-door occupations. In Paris, he thinks, there are fewer of the latter than of the former class. To me it is absolutely incomprehensible how so sensible a man as Grisolle can assert that, "considérés d'une manière générale"—I quote the original expression—tradesmen who occupy confined apartments are two and a half times less subject to pneumonia than those who work in the open air, or that women who live out of doors are much more predisposed than others. A greater contradiction between premises and conclusions than this can hardly be imagined.

The reports which Hannover has published for Copenhagen,[2] show that there is no apparent difference in the trades between an out-door or an in-door life; but he is very positive that such a difference does exist in the case of bronchitis.

Women are attacked by pneumonia somewhat less frequently than men. This point is more difficult to establish than it appears to be. Hence the contradictory statements of writers. The reckoning is often made by saying that for x men admitted into the hospital with pneumonia, there are y women, and the percentage is $\frac{x+y}{y} = \frac{100}{z}$. According to this plan the

[1] L. c., p. 112 et seq.

[2] Deutsche Klinik, Jahrg. 1861.

quantity *z* indicates the percentage of the women received into the hospital with pneumonia, as compared with the whole number of cases of this disease. But we forget to ask whether the women come to the hospital in as large numbers as the men. In every large city it is of course difficult to answer this question, because a very large part of the male population belong to the class of journeymen, day-laborers, etc. The floating female population is much smaller in comparison, and therefore the male population will avail themselves of the hospital in far greater proportion than the female. The fallacy of the above mode of reckoning the percentage is thus clearly shown; for if the male admissions number 200, and the female only 100, the reason may be that the female population, which resorts to hospitals, is only half as large as the male. This indeterminate quantity enters into all the hospital statistics.[1] We shall come somewhat nearer to the truth if we compare the number of both sexes ill with pneumonia with the number of admissions of each sex. If the numbers are sufficiently large, then the unknown and perhaps very important quantity in the problem is this: What proportion of each sex is taken care of outside of the hospital? This proportion will, of course, vary considerably in different places. I give below an illustration, on a large scale, of the differences between the two modes of reckoning.

In the Vienna General Hospital, during the years 1858–70 inclusive, there were 7,942 cases of pneumonia admitted, of whom 5,467 were men, 2,475 women. The relative percentages were 68.84 men, 31.16 women; or, expressed in another way, 45 women to every 100 men. Now the total admissions for that period were 299,929, of whom 188,273 were men, and only 111,646 women. Of the male admissions 2.9 per cent. were cases of pneumonia; of the female, 2.22 per cent. The proportion of women to men was therefore 77 to 100. These figures speak for themselves.

Nor can the mortality tables of the whole population be used, as they stand, for deciding the question of the relative frequency of pneumonia in the two sexes, because the percentage of deaths from this disease is greater among women than among men.

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[1] Compare the report of *Ziemssen*, which shows that in different hospitals the statistics vary upon this point from 16 to 60 per cent.—*Deutsche Klinik*, l. c., p. 60.

The data which Ziemssen gives show that out of 161,640 fatal cases of pneumonia 72,107 were women, 44.5 per cent.; 80 women to 100 men. If the polyclinics furnished statistics sufficiently large, we might, perhaps, with their aid, arrive at a definite result. I will mention, without ascribing much importance to the fact, that in the Würzburg polyclinic, among 565 cases of pneumonia, there were 85 women to every 100 men; in the Kiel polyclinic (200 cases) the proportion was 75 : 100.

Whatever method of reckoning we adopt, unless it be the impossible one which I first mentioned, we shall always find *that women possess a certain immunity in comparison with men; but that this is much less than it is stated to be by most writers.*

It is a more commonly received opinion that women are less often attacked by pneumonia, because they are not so much exposed as men to the influences of the weather.

In an agricultural population the women are without doubt more exposed than the same sex living in cities, as is shown by the relative frequency of pleuritis under these two conditions. In London only 41.3 per cent. of the fatal cases of pleurisy were women, while in the rural districts the number ran up to 60.9 per cent. In regard to pneumonia, however, the lists of mortality from this disease in England show how unfounded is the old idea upon this subject. The mortality for women is as follows:

Population wholly rural	41.2	per cent.	women.
Twenty-five cities of England (excluding London)	44.4	"	"
Manufacturing population	45.5	"	"
London	46.4	"	"

Irish statistics give a similar result.

The disposition on the part of the female sex to this disease is thus seen to be positively increased by an in-door life. We cannot, therefore, safely regard the nature of their occupation in life as the chief cause of their immunity.

The *age of the individal affords no protection from pneumonia.* Childhood and old age, adolescence and full maturity are all subject to its attack.

In regard to the proportion in whch the different classes of age suffer from this disease, we have at present no certain information. The difficulties in diagnosis are the special cause of embarrassment. Even a physician of limited experience can

easily recognize pneumonia in adults, but in *young children* this is by no means an easy matter. Every one who has seen the mistakes of his assistants in the practice of a large polyclinic, will confirm this statement. Many children, who are reported to have died from "teething," "worms," "convulsions," etc., have really gone to their graves with an undiagnosticated pneumonia. In the *aged* also the symptoms are often so latent that the disease may very readily be overlooked.

The statistics of hospitals often lose much of their value from the fact that old persons and young children are not admitted. It is to be taken into the account, also, that persons who have become settled in their own homes do not so frequently seek the care of public institutions when ill with a disease of such short duration as pneumonia, as when suffering from more chronic affections. This is shown by the fact that, for example, in Kiel, where a clinic and polyclinic have for years been in active operation by the side of each other, the proportion of cases of pneumonia admitted to the clinic and polyclinic respectively, was one to four. With typhus fever it is altogether different; here the number of clinical was double that of the polyclinical patients.

Nor can the statistics of mortality be used as they stand, because, since the power of resistance to the disease varies with the different periods of life, we cannot infer the number of cases of illness from the number of the deaths.

The following table [1] gives us an approximate idea of the relative mortality at different ages, although I suspect that catarrhal pneumonia, severe bronchitis, etc., have not been excluded.

Out of 1,000 deaths from all causes, at every period of life, in the whole population of England, the mortality from pneumonia (averages for two years) was as follows:

0– 5 years.	.....	95.1 per mille.	
5–10 "	.....	38.9	"
10–15 "	.....	23.1	"
15–25 "	.....	27.6	"
25–35 "	.....	29.9	"
35–45 "	.....	35.4	"
45–55 "	.....	37.1	"

[1] Taken from *Oesterlen*, l. c., p. 570.

55–65	years	35.4 per mille.
65–75	"	29.5 "
75–85	"	18.2 "
85–95	"	10.6 "
Over 95	"	5.4 "

This compilation shows that the first five years of life suffer the most severely, almost one-tenth of the deaths occurring from this disease. From the fifth to the seventy-fifth years the ratios do not exhibit very marked variations. In the records of deaths in Geneva, the mortality is stated to be much greater in the more advanced years; between forty and fifty years it rises as high as 101 per 1000. Whether this difference is due to diversities of climate, we are unable to decide.

The table below, although including but few cases, gives the statistics of morbility from pneumonia at different ages among 200 admissions to the polyclinic at Kiel:

0– 1	year	4.5 per cent.
1– 5	years	31.5 "
6–10	"	19.0 "
11–20	"	8.0 "
21–30	"	6.5 "
31–40	"	7.0 "
41–50	"	7.5 "
51–60	"	7.0 "
61–70	"	5.0 "
Over 70	"	4.0 "

The parallelism between this table and the statistics of mortality for England is obvious, although I have had to deal with small figures. Of course a morbility of 4 per cent. among persons over seventy years of age is much greater for this class than a percentage of 8 for persons between eleven and twenty years, because among the latter the number of individuals is much greater than among the former. In order to be strictly accurate, we must of course know the number of persons living at each age.

In opposition to the generally received opinion, it may be safely asserted that *persons in the prime of life are not especially exposed to pneumonia.*

The mistake on this point has arisen from a more or less exclusive use of hospital statistics. Take, for instance, those of the Vienna General Hospital. Out of 6,950 cases of pneumonia no less than 2,423 were between sixteen and twenty-five years of age. At first sight this fact seems to indicate a marked predisposition in persons of this age, whereas the true explanation is, that they came to the hospital because they had not been able to make homes for themselves, and were obliged to seek public assistance even for a brief illness. Grisolle has, more than any other

writer, subjected these statistics to the most searching criticism, and hence his views, which differ widely from those usually entertained, are well worth reading.

It is a very old belief that men of *strong constitution* are especially liable to be attacked by pneumonia. The fact is otherwise; a strong constitution protects against the disease. It is sufficient to refer to the large number of cases already mentioned as occurring in prisons, where the influences which weaken the body and diminish its powers of resistance to disease are so unusually active. We have additional evidence in the frequent occurrence of pneumonia towards the end of cachectic, carcinomatous, diabetic, and other diseases. On the other hand, when we consider the rarity with which the sea-faring population is attacked, it is very evident that a robust habit of body does not of itself predispose to pneumonia. Other writers, who have devoted special attention to this point, express the same opinion. Dietl found only 18 per cent. of previously healthy persons among 750 cases of pneumonia. At a time when the science of diagnosis was so imperfect that pneumonia was recognized only in persons of a vigorous age, it was natural to suppose that the disease did not occur in the feeble; but nowadays the belief finds few supporters. The frequency of croupous pneumonia during the course of severe acute infectious diseases might be adduced as collateral evidence. The complication occurs, as is well known, when the organism has already been considerably weakened by the preceding disease, and this is the explanation, in my opinion, of the greater disposition of these patients to pneumonia.[1]

It may have been a mere coincidence, but I can recall only a few isolated cases of croupous pneumonia occurring in my typhoid patients who were treated hydropathically.

Pneumonia rarely occurs in *pregnant women*, but when it does, miscarriage frequently results.

Among the 2,475 women treated in the Vienna Hospital from 1858 to 1870, 43 were pregnant. If we deduct 611, who were above fifty years of age, and 24 under thirteen, there remain 1,842 of a child-bearing age; of these only 2.3

[1] This is also the opinion of *Hugo v. Ziemssen;* compare Pleuritis und Pneumonie im Kindesalter, p. 154.

per cent. were pregnant. It is very evident that among the women in a large city admitted to a hospital the pregnant will show a larger percentage than this. Of the 43 pregnant women, the majority, 25, miscarried.

All writers agree upon this point.

One attack of pneumonia increases the disposition to a recurrence. This statement is probably correct, but it is not positively proved by sufficiently numerous observations.

Grisolle gives some data which are more accurate. Among 175 cases of pneumonia examined with satisfactory care, there were 54 who had already had one or more attacks, some as many as eight. Grisolle cites from Chomel cases with ten attacks, from J. P. Frank with eleven, from Rush with even twenty-two. In 35 cases the original seat of the pneumonia was ascertained. The recurrent affection occurred twenty-five times in the lung previously attacked. The above statement is thus seen to be confirmed by statistics, and in fact accords with our daily experience.

It is a current opinion that the prosperous suffer less from pneumonia than the poor; but this statement should not be accepted without further examination. This is but another instance of the readiness with which we are decided by appearances. Every busy physician with a mixed practice treats more cases of pneumonia among the laboring classes than among civil officers or business men, and hence retains a more vivid recollection of the cases among laborers than among the educated. It is evident that the upper classes are far outnumbered by the lower, and that, for example, among the 10,000 persons who rule England there will be much fewer cases of pneumonia than among the coal-miners. But still there are facts which certainly seem to show that persons with favorable hygienic surroundings (and in general this is doubtless the condition of the wealthy, as opposed to the poorer classes) are less exposed than others to attacks of pneumonia. This is strikingly shown by the tables of mortality for the English army. At all the military posts the mortality is less among officers than among the rank and file.

During the longer periods of from ten to twenty years, the ratio was 12 deaths per 1,000 among the rank and file, and only 5 per 1,000 among the officers.

This marked difference is probably due to general hygienic conditions, because the age and kind of occupation is the same in both classes, and both have the same early medical treatment.

Of similar significance is the fact, that in the English *army* and *navy* pneumonia has of late years been much less frequent, owing, doubtless, to improvements in the commissariat and clothing; in a word, to better hygiene.

In England the yearly mortality per 1,000 was:

1830–36	0.83 cavalry.	2.31 infantry.
1837–46	0.44 "	0.91 "

A more or less considerable reduction in the frequency of pneumonia has also been noticed at all the foreign stations.

It is stated by veterinary physicians that true croupous pneumonia often occurs in *animals*, generally *horses* and *cattle;*[1] in fact, the nature of the symptoms and the lesions found after death, leave no doubt upon this point. The disease is said to frequently prevail as an epidemic.

In discussing the question of the *epidemic* occurrence of croupous pneumonia, it is important in the first place to come to a clear understanding of terms. There is no reason to suppose that the so-called "typhoid pleuropneumonia," the "pleuritis pestilens," or the "Alpenstich" belong to this disease. These affections, which appear in epidemics of greater or less extent, and exhibit characteristic constitutional symptoms in addition to the pulmonary lesions, belong to the group of pestilential and typhoid diseases. The lesions of the lungs, and, perhaps, also, of the pleura and pericardium, together with the occasional occurrence of bloody expectoration, the nature of which has not been carefully observed, are all that these affections possess in common with croupous pneumonia. If we include them under the name of pneumonia, we have as good a right to include those hemorrhagic forms, which we sometimes see nowadays in severe small-pox, scarlet fever, etc. Nor can we consistently exclude the epidemic disease known as the "black death," when the lesions are localized especially in the lungs. Such a view, it seems to me, tends to confusion rather than clearness. For this reason I separate the so-called "typhoid" pneumonia from the croupous, and shall consider only the epidemic occurrence of the latter. *Croupous pneumonia belongs to the per-*

[1] *J. E. Veith*, l. c., II. Bd., p. 61.

manent diseases—the endemic class; it recurs in annual cycles. In a certain sense we are justified, on the continent, in speaking of May epidemics of pneumonia, just as in places where typhoid fever is a fixed affection we regard its regular annual increase as an epidemic. Individual years, when compared with each other, exhibit a varying frequency of pneumonia as well as of typhus, malarial, and other endemic diseases.

The mortality during a period of eleven years in England, averaged 1.25 per 1,000 inhabitants; the maximum during any one year was 1.44, the minimum 1.11. The variations were even greater in Ireland. During a period of nine years the average of the total mortality was 0.27, the maximum 0.41, the minimum 0.14 per 1,000. If we regard the average as a unit, and compare the variations with this standard, the "extreme variation" for England would be 26.4 per cent., for Ireland 100 per cent.[1]

In this sense it is certainly allowable to speak of epidemics of croupous pneumonia, but this is only a partial statement; for besides the more or less severe outbreaks of the disease, there is everywhere to be noticed a certain continuity during the intermediate periods.

Some years show a maximum frequency of pneumonia, others a minimum. The year 1847 showed the highest maximum, then follow 1838, 1840, 1842, 1844, etc. The minima occurred in 1836, 1839, 1841, 1845–6, etc.

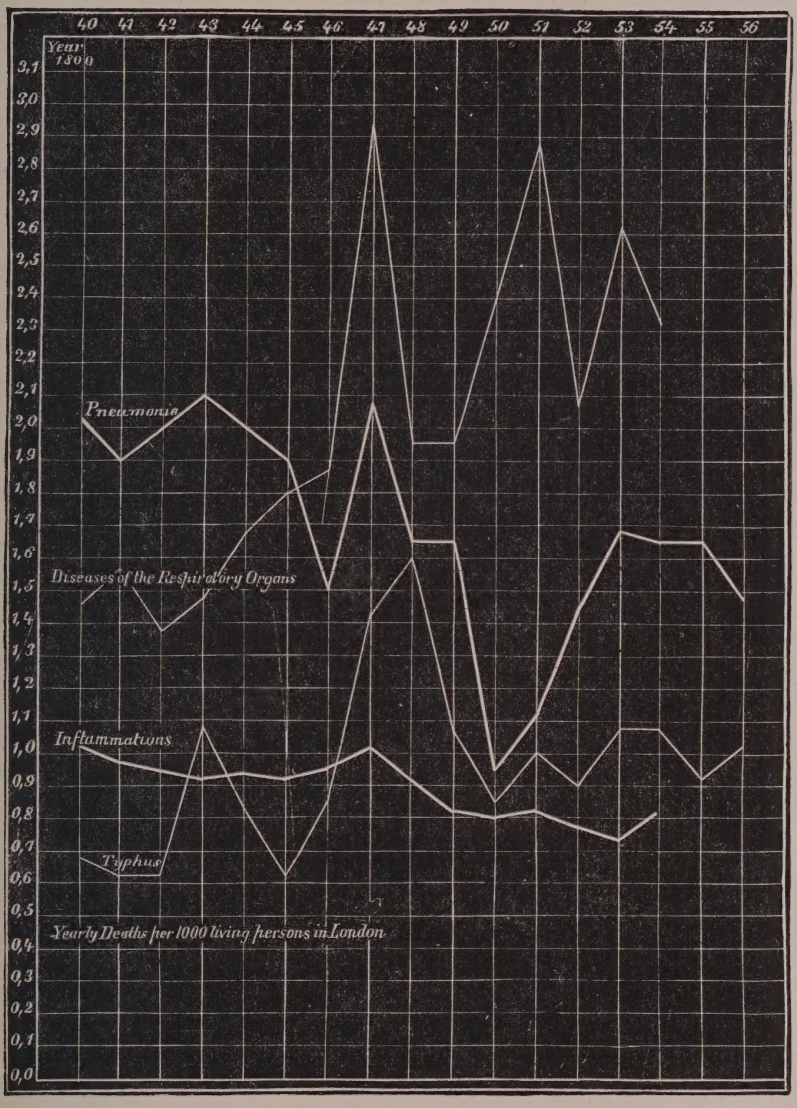
In 1847, sixteen European towns out of nineteen, in which accurate statistics were kept, showed a high point, and twelve their absolute maximum (the period of observation extending over twenty-one years). In 1848 four others reached their maximum, which was, however, somewhat lower. In America pneumonia is stated by physicians to have been unusually frequent during the year 1847. On the other hand, the years 1845 and 1846 were characterized by a general decrease in the number of cases.

From the mortality reports of London, Ziemssen has made a comparison of the diseases which coincide in point of time. This comparison possesses great etiological interest, and I give below the chief points, premising that it embraces the period from 1840 to 1856.

1. The *inflammatory diseases* (including under this general name pericarditis, pleuritis, laryngitis, cephalitis, hepatitis, peri-

[1] *Ziemssen*, Prager Vierteljahrschrift, v. 1. c., p. 11.

tonitis, gastritis, and enteritis) present annual ranges which are very different from those of pneumonia, and vary but little in frequency from year to year.



2. The diseases of the *respiratory organs* (excluding pneumonia, tuberculosis, pertussis and croup) occur more frequently, but do not always coincide in point of time with pneumonia.

3. There is a remarkable coincidence between the typhus and the pneumonia years.

The foregoing curve, drawn in accordance with Ziemssen's report, shows these relations in detail.

It is impossible to reconcile these facts with the supposition that there is a close causal connection between pneumonia, on the one hand, and "inflammatory diseases" in general, or the affections of the respiratory organs, on the other. How little real importance there is in general theories as to the essential nature of diseases, may be shown by the following facts. Of the different "type" theories of disease, which prevailed and rapidly succeeded each other during the close of the last and the early part of the present century, the theory of an "inflammatory type" maintained its sway from 1811–1820. This was preceded by the "adynamic-nervous" theory, and followed by the "gastric-bilious." Now, a careful analysis of the annual reports of the Child's Hospital in Vienna, from 1796 to 1819 (total admissions 127,408), shows that during the prevalence of the "adynamic-nervous" type (1796–1810), pneumonia formed 2.9 per cent. of all diseases, and during the prevalence of the "inflammatory" type (1811–1820), 2.86 per cent. The maximum 3.48 per cent. occurred during the quinquennium, 1801–1805, which belonged to the "adynamic-nervous" period.

It is customary to enumerate as *exciting causes* many things, which may, it is true, excite an inflammation of the lungs, but not a croupous pneumonia. To this class belong injuries to the chest by contusions, perforating wounds, or those caused by foreign bodies which are drawn down into the bronchi, and excite pulmonary lesions. I shall at present content myself with showing that no injury of this kind can produce a true croupous pneumonia.

A frequently quoted case of "traumatic pneumonia," related by Morgagni, illustrates the lack of critical judgment with which observations are sometimes made. A boy fifteen years of age fell while wrestling; his opponent sprang upon him, and knelt upon his chest. The patient complained of pain at a spot (the last dorsal and first lumbar vertebræ) where no important injury was perceptible. He felt unwell from the start, after a few days became feverish, and symptoms of pulmonary inflammation appeared. Death on the tenth day after the accident. The autopsy

showed that the internal injury at the contused point was as slight as the external one. The upper part of the right lung was found to be inflamed.[1] The case is now quoted as proving that pneumonia may arise from traumatic causes.

In what proportion of cases the attack of croupous pneumonia arises from the *chilling of the body*, has long been a matter of dispute. Opinions upon this point range from one extreme to the other. The older physicians were accustomed to regard the chilling of the body as a *conditio sine quâ non;* latterly the tendency is to deny it any influence whatever. The etiological conditions, which have already been considered, seem to me to confirm the view that there is no absolute causal connection. Every physician of large experience knows, without reference to statistical data, that pneumonia can occur without a previous chilling. It is quite another question whether such chilling is a frequent exciting cause. For exact figures and statements upon this point we are again indebted to Grisolle and Hugo v. Ziemssen.

A careful examination of cases showed that 45 out of 205 patients sick with pneumonia ascribed their illness to exposure to cold. Some of them had been exposed, while heated, to draughts of cold air, some had had their clothing wet through, and others had become chilled by drinking very cold liquids. In 34 cases Grisolle ascertained the time which intervened between the exposure and the commencement of the illness. In 18 persons the prodromata of pneumonia, such as general malaise or local symptoms of the disease, occurred during the exposure, or a few minutes afterwards; in 11 the interval was from two to three hours, in 4 from one to two days. Among 186 cases there were 10 in which Ziemssen ascertained that the chill was the exciting cause. The interval between the exposure and the outbreak of the disease varied from half an hour to sixteen hours.

These facts are sufficient to show that *a chill is by no means a frequent cause of croupous pneumonia*, and the attempt of Lebert,[2] in an indirect manner, to restore it to its old influence will probably prove as unsatisfactory as his hypothesis that the disease is excited by obstructed perspiration. Hirt[3] states that the continued inhalation of dust not only favors the occurrence

[1] *Morgangi* de sedibus, etc., Lib. II., Ep. XX., 28.

[2] Klinik der Brustkrankheiten, Bd. I., p. 713.

[3] Staubinhalationskrankheiten, p. 15 et seq. Breslau. 1871.

of croupous pneumonia, but even directly produces it. In disproof of this opinion I refer to Merkel's statements in his article on this subject.[1] How little convincing Hirt's statistics are, is best shown by his own tables. Here is an example. Among the persons who inhale mineral dusts, carpenters are said to be attacked by pneumonia in the proportion of 6.9 per cent., masons 4.4 per cent. There can be scarcely a doubt that masons are, as a class, much more exposed than carpenters to dust. The percentage of cases among coal-merchants is 14.4 per cent., among coal-miners only 4.7 per cent.!

Severe exertion of the lungs, as in singing or playing upon wind instruments, does not produce any special disposition to pneumonia.[2] The statement that pneumonia is produced by the bite of serpents, especially the rattlesnake, appears to be mythical.

PATHOLOGY.

Outline of the Course of the Disease.

There is no disease which presents such a variety of aspects as croupous pneumonia. The only constant element is the anatomical change in the lungs, which can be discovered only by a careful physical examination, and by the presence of a single functional disturbance, the disproportion between the frequency of the respiration and that of the pulse. All the forms of the disease are connected together by the local affection, which ought to be demonstrable; the etiological factor, which so often turns the scale in acute affections we shall not stop to consider here. If we bear in mind also that many cases of pneumonia present very few of the symptoms which the text-books press into the foreground of their vivid pictures of the fully developed disease as it occurs in vigorous adults, we shall understand why pneumonia so often remains undiagnosticated. The physician who has a chest of drawers made with the labels

[1] This article is to be found in the first volume of the German edition of this Cyclopædia. This volume is omitted from this edition on account of its decidedly local character.—TRANSLATOR'S NOTE.

[2] *Grisolle*, l. c. p. 155.

"gastric fever," "rheumatic fever," etc., who has a large practice among children suffering with "worms" or "convulsions," and who loses many of his patients from "senile debility," may rest assured that many of these cases have been pneumonia. Usually the respiratory affection predominates; sometimes, however, the symptoms extend in all directions and the whole body is involved. At one time the invasion is violent, the brain is chiefly affected, and death ensues frequently in one or two days; at another there is a lingering illness, the only important lesion seems to be with the nutrition, and the patient sinks from exhaustion through the gradual extinction of the vital functions; in other cases there is a struggle for breath, a battle of the respiratory organs apparent to the dullest eye, and the fatal result is inevitable. The duration of the disease is as variable as the symptoms. There are undoubtedly cases of pneumonia which terminate within twenty-four or thirty-six hours; then again there are others in which the disease lingers for months in so latent a form that it is impossible to say when the primary affection ends and the sequelæ begin. Between these two extremes there are all possible transitions. When we consider that croupous pneumonia attacks persons of all ages, we can understand how this endless variety of individual elements should produce an ever-changing picture of the disease. One link of this chain removed from its natural connection we describe under the name of croupous pneumonia. For the purpose of description we shall commence with the typical disease, but merely for convenience, it must be remembered, and not from necessity.

Pneumonia generally begins suddenly with a chill. The patient, who has hitherto been quite well, is struck down as if by lightning out of a clear sky. He awakes in the middle of the night, his teeth chatter, a shivering seizes him, his chest heaves with the difficulty of respiration; then follow quickly pain in the side and a short paroxysmal cough, attended with severe pain, but no expectoration. He presses the painful spot with his hand, in order to get relief by the pressure. Headache, vertigo, heaviness in all the limbs. Soon the face becomes flushed, the cheeks appear somewhat cyanotic, the alæ nasi

move, and the countenance exhibits a somewhat anxious expression. He sits half erect in bed supported by pillows, and in vain attempts to suppress the convulsive paroxysms of cough. This effort gives the cough a peculiar character; it sounds as if interrupted, hoarse, and harsh. The skin is burning hot, and dry, the pulse increases in frequency, and the patient suffers from intense thirst. Thus within a few hours the disease may have already reached its height.

At other times the pneumonia is developed more gradually. For several days the patient feels unwell and is very chilly, then the pneumonia is ushered in by one or more marked rigors, and the other characteristic symptoms. The initial chill may be absent, and in its stead the scene opens with convulsions and complete loss of consciousness. Occasionally the disease progresses to its full development without the presence of any objective symptoms whatever. If there be a chill, the illness may be dated from this point; in other cases the chronology remains uncertain, for complete infiltration may take place without any complaint on the part of the patient. When croupous pneumonia attacks a person already feverish, the temperature does not usually undergo any important change. This is particularly the case when the fever is of a high grade, and very noticeably when it is associated with local derangements of the central organs. So also in cachectic conditions the thermometer gives us no certain indications of the supervention of pneumonia. In the absence of a chill, the time can be accurately computed only when the patient, having previously been in good health, can fix with certainty the moment when the change commenced. This will be possible only when the onset of the disease has been acute, but in this case the chill will almost always be present.

Another initial symptom of frequent occurrence is vomiting. This may take place either before or with the chill, and is generally repeated. Hence the frequent complaint of the patient that he has vomited bile.

Prodromata are not so rare as many suppose. Grisolle, who gives us positive information upon this point also, states that he has found premonitory symptoms in fifty out of two hundred and five cases of pneumonia. Of these patients forty-four complained of general malaise, the pneumonia developing gradually without the

occurrence of any new symptom; three stated that the indisposition was preceded by a cold, and one by intoxication. The other two felt unwell and complained of heaviness in the limbs and anorexia, one for three, the other for six days previously; after a chill, the symptoms of pneumonia developed immediately. The duration of the prodromata varied between a few hours and several weeks. In the latter case it seems to me that the symptoms can hardly be regarded as prodromata. Grisolle thinks that a prodromic stage is more common in persons between thirty and forty, and fifty and sixty years of age, but he very properly avoids insisting upon this point. After the age of seventy pneumonia is said to develop, as a rule, without premonitory symptoms; but my own experience is opposed to this view. Hugo v. Ziemssen[1] and myself agree in the opinion that a prodromic stage rarely occurs in hearty children.

At the commencement of pneumonia the physical signs are uncertain and ambiguous. For at least twenty-four hours after the chill the percussion note is generally unchanged, and the same is true of the vocal fremitus. Auscultation reveals at the affected point a somewhat diminished intensity of the vesicular murmur, sometimes associated with a slightly rough sound. If large or small, but not sibilant catarrhal râles are heard in this stage upon one side only, they indicate the locality of the disease.

Until the disease is fully developed the general symptoms remain much the same. Fever, with a temperature ranging from 104° to 105°, falling from 0.9° to 2.7° in the morning, and rising again in the evening to an equal amount; pulse 100–110; respiration 40–50 per minute; headache, exhaustion, thirst; in short, all the symptoms of the febrile condition. The skin, which is at first generally dry, usually becomes moist about the third day, at least temporarily. The cyanosis of the cheeks, the anxious expression of the face, the respiratory movements of the alæ nasi continue; the patient speaks in broken sentences, and in a low tone of voice. The cough occurs in paroxysms, maintains its peculiar character, and is always accompanied by pain. When the pain becomes intense, the patient continually tries to restrain the affected side by the pressure of his hands, by binding it, or by lying upon it. The sleep is often more or less disturbed by the frequent paroxysms of cough. Usually the accessory muscles of respiration are called into play, and the trapezii

[1] Pleuritis und Pneumonie im Kindesalter, p. 166.

and sterno-cleido-mastoid muscles are seen to be tense; less frequently the patient uses the muscles which are liberated by the fixation of the arms. The sputa, which at first are mucous, and afterwards slightly purulent, become more and more bloody. The streaks of intimately mixed blood and mucus, which are first seen upon the sputa, become gradually broader and broader, until they finally extend over the whole of the expectorated mass. The color of the sputa is reddish brown; they are mixed with air-bubbles, and cling firmly to the sides of the vessel containing them. There are generally also to be found muco-purulent masses, such as occur with every catarrh. The quantity of the expectoration is almost without exception small. Herpes vesicles frequently appear about the mouth, usually only a few, but sometimes in great number. The urine is diminished in quantity, dark colored, of high specific gravity, and, on cooling, deposits a moderate sediment of brick-dust urates. Micturition is in many cases more frequent than usual, and accompanied by slight scalding. The bowels are usually constipated. The physical examination shows, in progressive order, a slight impairment of the percussion note, which has also, very frequently but not always, a moderately tympanitic quality, and moreover, at this time, a diminution of the pectoral fremitus. The respiration is feeble, indefinite, and harsh, with perhaps a few crepitant râles. Thus up to the fifth or sixth day the local signs may not be clearly defined, especially when the auscultatory signs of infiltration are disguised by a severe peripheral bronchial catarrh. Usually, however, up to the third day after the chill the percussion note over the affected part becomes dull, the vocal fremitus increases, the respiration becomes of a blowing character, the expiration is clearly perceptible, and crepitant râles are heard mixed with large and fine bubbling sibilant rhonchi. Bronchophony now makes its appearance. From a point, which is usually small at the start, these signs radiate in a few days into the surrounding parts. Almost always towards the end of the attack we may observe in the affected portion of the lung the whole scale of changes from their beginning to their complete development. If the cough have ceased for a time, we may perhaps hear nothing upon auscultation; but the characteristic

signs of infiltration return, when the patient is made to cough violently. At any time up to the height of the disease, if the signs of condensation diminish, and gradually return to their earlier character, we may infer a resolution of the inflammation. On the other hand, fresh portions of lung may become involved when the disease has reached its height. In rare instances a complete infiltration can be discovered by physical signs within the first twelve or twenty-four hours.

Sometimes after a few days, generally towards the end of the first week, an abatement in the symptoms takes place. This is first shown by the pulse. The arterial tension diminishes, the pulse becomes softer, and at the same time slightly irregular. The pulsations of the heart do not succeed each other with so much regularity as before, its impulsive force varies in amount, and the pulse in fulness and strength. This condition may be observed within twelve or fourteen hours before the beginning of the crisis, sometimes a few hours earlier. It may also be noticed at this time, sometimes a little later, that the cheeks are less congested, and that the patient is perspiring more profusely, especially on the forehead, where the sweat stands in drops. Locally no evident change may be detected; even the temperature keeps at its former height, or may increase a few tenths of a degree. Nor do the feelings of the patient reveal the improvement which is impending, but relief is experienced from the moment when the thermometer sinks rapidly. Within from four to sixteen hours the temperature becomes normal, the pulse and respiration diminish in frequency, and all the symptoms abate; at the same time the whole surface of the body usually breaks out into a warm, free perspiration. A deep and continuous sleep follows, and on awaking the patient feels comfortable, enjoys his food with a keen relish, and convalescence commences. The cough becomes looser and easy, and is painless, or attended with but slight distress. The purulent or mucous expectoration, if it still continue, gradually diminishes. The urine, which is still not much increased, throws down a more copious deposit than before. For the first twenty-four hours after the occurrence of the crisis there is not much change in the physical signs, except that the crepitant râles are heard over a

wider extent; the bronchial breathing and sibilant rhonchi still remain. In the course of from four days to several weeks the local signs gradually disappear. Usually, however, the percussion note is diminished in intensity for several months afterwards over the original seat of the pneumonia.

In place of the above-described crisis, the attack may terminate by lysis. The difference is rather quantitative than qualitative, one process requiring only hours, the other days. The gradual fall to the normal, which is noticed in all the symptoms as well as in the temperature, requires more than one day for its completion. There may even occur temporary exacerbations, with renewed elevations of temperature, interrupting the general fall. In other respects this mode of termination is very similar to that by crisis, except that these interpolated exacerbations of the symptoms, which have not yet wholly abated, give it an appearance of irregularity. In about thirty-six or forty-eight hours the return to the normal condition appears to be completed. Variations from this, which is the usual course, are sometimes noticed. On the day of the crisis indications of defervescence occur; but these soon give place to an exacerbation with the symptoms of the height of the disease, and convalescence does not begin until towards the middle or the end of the second week. Or the acute symptoms subside, but the temperature keeps above the normal, the signs of pulmonary disturbance do not completely disappear, and the disease afterwards runs a chronic course. Or it may happen that the fever and local affection continue with scarcely any manifestations of a crisis, and some acute complication is developed, which is usually excited by the local affection, or proceeds directly from it. Finally, an interval of almost entire absence of local symptoms may elapse between the termination of the pneumonia and the occurrence of an inflammation of a neighboring organ secondarily to the pulmonary inflammation. Slight febrile movements are in this case the only indication that there is still something wrong.

Death may take place early, sometimes within a very few days, with intense fever (about 106°), severe cerebral symptoms, convulsions, and delirium, reminding one very much of a violent

meningitis. The local symptoms may be quite latent, but will seldom be entirely overlooked if examined for with care. Death occurs from exhaustion of the heart, generally towards the end of the first week, but sometimes earlier. The cyanosis increases, the dyspnœa becomes more urgent, the shallow respirations more and more frequent, and the pulse more rapid, smaller, and sometimes irregular. The temperature of the trunk is considerably increased, while the face and extremities are cool, and covered with a profuse perspiration. The tip of the nose first becomes cold, then the hands, and finally the feet. The deep red color of the cheeks becomes more and more bluish, and at last assumes a deep yellow tinge, as in heart disease. The sensorium is obtunded, and the patient lies in a low delirium, talking little, and motioning with his hands. He is easily aroused for the moment by being spoken to. He understands what is said to him, but his replies are so indistinct, low, and frequently interrupted, that it is very difficult to ascertain their true meaning. The skin becomes anæsthetic, so that a hypodermic injection excites no pain. The respiration is gasping, and accompanied by coarse, moist râles, which can be heard at some distance. The fluids given to him can no longer be swallowed, and, entering the larynx on account of imperfect closure of the glottis, excite feeble attempts to cough. The breathing becomes more and more feeble, and is interrupted by pauses of constantly increasing length; the radial pulse is no longer to be felt. Thus death closes the scene, generally several hours after the commencement of the urgent symptoms. In a few instances the patient, on suddenly rising in bed, falls back dead from acute exhaustion of the heart. In elderly persons also the final struggle is brief, the symptoms being those of rapid collapse. Sometimes, however, in these cases death ensues gradually. It is not very rare for the catastrophe to happen at the time of the crisis—the temperature sinks below 96.8°, the pulse becomes slow or even very rapid, and colliquative perspiration and œdema of the lungs take place. In a word, the patient dies in collapse, although the symptoms are somewhat different from those of the usual form. The complications and sequelæ present a character peculiar to themselves. This is not the place to describe them.

Convalescence is generally completed rapidly and easily, unless retarded by an unusual delicacy of constitution or by the prostrating nature of the treatment. It is only after a very severe pneumonia that the hair falls out, or much desquamation of the epidermis occurs; in a word, there seems to be no such profound disturbance of the whole constitution after pneumonia as is the rule in typhus. In a fortnight at most after convalescence begins, and very often in a much shorter time, the laborer, if he have previously been a man of sound constitution, is able to return to his work.

Formerly the practice was more common than now, in describing the course of pneumonia, to divide it into "stages." These stages—"engorgement" and "red and yellow hepatization"—are anatomical, not clinical, in their nature. It is a sufficient objection to such a division that the stage of "red hepatization" is always accompanied by "engorgement" of some portions of the lung. The "yellow" or "gray hepatization" may not occur at all, and if it does, the other two stages are generally present also. The division of the course of the disease into stages, certainly cannot be based upon the anatomical condition.

Outline of the Morbid Anatomy.

The principal lesions in those who have died of pneumonia are found in the lungs and pleura.

The body is generally well nourished, the rigor mortis well marked, and the skin cyanotic, with numerous livid spots. The muscles are dry and brownish red, sometimes partially of a pale grayish yellow color, as in typhus fever. The brain is slightly œdematous with serum, and much congested. The jugular veins and venæ cavæ are distended with blood. The liver, spleen, and kidneys are usually considerably congested. I have seldom failed to find the spleen enlarged by about one-half. Catarrh of the pelvis and papillæ of the kidneys is an almost constant lesion. In the intestines no changes are uniformly found, except here and there a slight enlargement of Peyer's patches and of the solitary follicles. When the chest is opened, the lungs generally collapse less than usual, and the non-infiltrated parts appear somewhat raised. These portions are generally œdematous, and contain variable quantities of blood; *i.e.*,

congested spots are distributed in the midst of tissue which is comparatively bloodless. A frothy fluid, mixed in many places with muco-pus, oozes from the cut surface.

The lesions found at the special seat of the inflammation are divided by pathologists into three stages. In the following description I have adopted substantially the words of Rokitansky.[1]

First Stage; Inflammatory Engorgement.—The lungs are of a dark red color, heavy, firm, and retain the depression caused by the pressure of the finger, showing that they contain little or no air, but fluid. On section the substance is found to be dense, the result of swelling of the tissue and infiltration with a bloody serous fluid. According to the degree of this condition the lung crepitates and floats upon water, because it still contains some air, or it does not crepitate, and sinks in water to the bottom. It is, moreover, easily torn, very moist, and exudes a bloody, serous fluid, which is sometimes frothy, sometimes not.

Stasis, especially when combined with œdema, presents the greatest similarity to this condition.

The inflammatory engorgement is distinguished from this condition by its color, which approaches a brownish red, and by the moisture of the parenchyma resulting from its infiltration by a brown or brick-red, thin, viscid fluid mixed with black grumous masses.

When the transition to the second stage takes place, a very adhesive, viscous, reddish-brown fluid is secreted. Then comes the exudation proper; and the *second stage—hepatization*—begins.

The lung is now, both externally and internally, of a dark brownish-red color, solid, but easily torn, no longer crepitates, and sinks in water to the bottom. On section the color above described is either uniform, or presents a marbled appearance, the black lung substance being seen as irregular spots, the pale red interlobular tissue as ramifications, the whitish bronchial and pulmonary vessels as longitudinal streaks or as islands. When the tissue is cut or torn, the surface has a characteristic

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[1] L. c., Bd., III., p. 48 et seq.

granular appearance. The granulation is uniform, and consists of isolated granules. Upon the cut surface there exudes, usually only on pressure, a brownish-red, opaque, bloody, serous fluid, mixed with blackish-brown and grayish-red flakes. This condition, which is known as red hepatization, after many transitional changes which imperceptibly succeed each other, passes into the *third stage proper*. The red-colored lung gradually becomes paler; the color changing first to a brownish red, then to a grayish-red and gray, and finally almost to a yellow (*gray hepatization*). The color is not uniform, particularly upon the cut surface; for while the grayish-red and gray, with here and there a shade of yellow, constitute the fundamental color, there are interspersed, at more or less regular intervals, the white sections of the divided vessels and the black substance of the lungs, giving to the whole the well-known appearance of granite. The granular texture still exists, and is even more conspicuous when the disease has run a tumultuous course. The consistence and red color diminish together. The lung, to be sure, feels tolerably firm, but it pits slowly on pressure, is soft, easily torn, and on section exudes a grayish-red, very opaque, flocculent, viscid fluid.

The transition into the *third stage*, that of *purulent infiltration*, is effected in the following manner: The decolorization into the yellow hue already mentioned goes on increasing, the granulation of the surface generally disappears rapidly, and is replaced by a purulent infiltration of the parenchyma. The lung is now heavy, and pits upon pressure. The cut surface is of a straw-yellow color, interspersed with the black of the lung, and from it exudes, in large quantity, a very viscid purulent fluid of a faint odor. The parenchyma is very soft, and tears upon the slightest pressure. If a piece of the lung be carefully pressed and washed free from pus, it will be seen that its substance has regained its spongy cellular texture.

The portion of the pleura covering the inflamed section of lung always exhibits changes at those points where the infiltration has extended to the surface. These changes consist at first of great injection of the blood-vessels, and small subpleural ecchymoses with slight opacity of the membrane, and after-

wards increase to a considerable thickening of the pleura, pseudo-membranous deposits, and a moderate amount of effusion into the cavity of the chest. From the point of the pleura pulmonalis first affected the inflammation extends in all directions, even to the pleura costalis. The pericardium and peritoneal covering of the liver sometimes become involved. In pneumonia of the upper lobes a pleuritis may be developed over the diaphragm, without there being any appearance of continuity between the two lesions. This is, however, a rare occurrence.

The bronchial glands are generally congested and swollen.

The bronchi present a variety of changes, especially in the terminal branches. During the first stages the mucous membrane is reddened and swollen; later it becomes paler, and almost always the tubes contain an exudation, which is first reddish, then whitish, and ultimately breaks down into pus. The vessels are frequently plugged by coagula of a similar appearance.

The heart sometimes undergoes structural change; its muscles are of a light grayish-red color; the right ventricle is filled with coagula, and the left empty.

Croupous pneumonia is, anatomically considered, an acute inflammation of the alveoli and bronchioles, in which a fibrinous exudation is poured out upon the free surface of the mucous membrane and there coagulates.

The *histological* examination gives the following results: *Inflammatory engorgement.*—"All the blood-vessels of a large part of the lung are distended with blood. The capillaries project far into the cavity of the alveoli, and evidently narrow it. The effusion which first appears consists of an albuminous viscid fluid, and this is succeeded by exudation and extravasation. Here and there, especially in the connective-tissue septa and under the pleura, there are found small punctiform extravasations of blood." Thus far Rindfleisch,[1] whom I have been obliged to follow, because I have no views of my own to give.

Red hepatization.—The alveoli are filled with a fine network

[1] L. c., p. 386.

of fibrine, in which are imbedded numerous white and a few red blood-corpuscles. The epithelium is intact. Here and there are found some cells with several nuclei. The pulmonary capillaries are slightly distended, and their cavities completely filled with blood-corpuscles, which are closely packed together, and have become polyhedral by being flattened against each other. The interstitial septa are slightly swollen.

Solid coagula, quite similar to those filling the alveoli, are also found in the bronchi leading to the inflamed part, and may extend as far as tubes measuring one-fifth of an inch in diameter. In these bronchial coagula also there are present numerous white and a few red blood-corpuscles.

The granular appearance of the cut surface, which even to the naked eye is so characteristic of croupous pneumonia, is produced by the alveoli and bronchi acting as moulds in which the fluid exudation coagulates.

The more delicate changes of this stage I have seen illustrated in the preparations of my respected colleague Schüppel. Although I entirely agree with Rindfleisch upon the essential points, I may be allowed to differ from him in regard to one subordinate detail. In my opinion the red color of the inflamed organ is not due chiefly to extravasated blood. In view of the relatively small number of red blood-corpuscles, which are usually found in the fluid upon the cut surface, I prefer to suppose that the color is produced by the distended blood-vessels, which are seen through the overlying more or less transparent exudation. This explanation is not intended to apply to the case where the exudation contains so much blood that it is more correct to regard it, as Rindfleisch does, as an extravasation.

The question arises, What is the source of the different constituents of the exudation—the red blood-corpuscles, the white or pus corpuscles, and the fibrine? There can be no doubt as to the red corpuscles, because they can come from no other source than the blood; and if we adopt Cohnheim's views, it will scarcely be denied that the greater number of the structures usually called pus-corpuscles have the same origin. But it may be questioned whether, in fact, all of the pus comes from the vessels. Buhl[1] denies that it does. From the fact that in many epithelial cells as many as from ten to twenty pus-corpuscles may make their appearance, he infers that the epithelium of the

[1] L. c., p. 25.

alveoli takes an active part in the formation of the pus. This fact, which Buhl[1] published some time ago, has been confirmed by others, but there is some difference of opinion upon this point. As the nucleus in the cells in question remains intact, the newly formed cytoid structures cannot have been produced by its division. Buhl therefore supposes that an endogenous free cell-formation takes place through a sort of fission process. If this were the case, this mode of formation would be the only instance of the kind in the fully developed organism. Volkmann[2] and Steudener oppose this view, and hold that the young epithelial cells are simply invaginated by the pus-corpuscles which have forced their way into them. Heller[3] and Friedländer[4] adopt the same opinion. It would ill become me to give my opinion in this matter, when even Virchow[5] reserves his own. I will only remark that in his first communication Buhl states that the lungs which he examined were passing from the red into the gray hepatization, a period when active inflammation has for the most part ceased.

It is far more difficult still to come to a positive conclusion in regard to the origin of the fibrine. Does it come directly from the blood, or have the tissues of the affected part any influence in its production? How great is this influence? These are questions of the greatest delicacy.

It has been ascertained beyond question by Cohnheim that an alteration in the wall of the vessel is a necessary part of the inflammatory process, and that the formed elements of the blood can pass through a wall which has undergone this change. If the corpuscles can escape, there is certainly nothing to prevent the passage of fluids, and in fact Cohnheim has demonstrated this also by experiment. The fluid, which is effused from the blood in inflammations, has been shown to possess different qualities, especially in regard to the facility with which it coagu-

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[1] Virchow's Arch., Bd. 16, p. 168.

[2] Centralblatt f. d. medic. Wissenschaften, Jahrg. 1868, pp. 257–8.

[3] Sitzungsber. der physical-medicin. Societät zu Erlangen. Sitzung vom 6. Mai, 1872.

[4] Untersuchungen über Lungenentzündung, p. 23. Berlin, 1873, Hirschwald.

[5] Cellularpathologie, 4. Auflage, p. 490.

lates. The cause of this difference is as yet unknown. "Of course the chemical properties of the fluid, which has exuded from the vessels, will have much to do with this point, and perhaps also the special way in which the walls of the vessels are changed may not be unimportant." [1] It is not impossible that the epithelial cells may also have a share in the process. Even if, like Wagner,[2] we refuse to acknowledge, without further evidence, that the croupous exudation is derived from a direct conversion of the epithelium, yet it must be admitted, as proved by the investigations of Wagner and Bayer,[3] that a peculiar change does take place in the epithelial cells during a croupous inflammation. On the other hand, no one has ever yet succeeded in artificially producing a croupous pneumonia,[4] although the genuine phenomena of inflammation are produced in the blood-vessels (Friedländer). The fact, so strongly insisted upon by Virchow,[5] that local differences influence the composition of the exudation, is probably applicable here. So far as I have examined the subject I know of no facts opposed to this view. In croupous pneumonia the lungs are acted upon by an irritation, which not only excites the ordinary lesions of inflammation, but also by its special influence upon the epithelium produces a coagulable exudation in the alveoli. This irritation no one has hitherto been able to induce by the ordinary means of exciting inflammation.

Cohnheim's well-known investigations furnish also a satisfactory explanation of the gradual change from the red to the yellow hepatization. I quote his own words: [6] "The diapedesis of the red blood-corpuscles out of the capillaries always takes place simultaneously with the extravasation of the white corpuscles, but of course with more difficulty than the simple fluid

[1] Cohnheim, l. c., p. 76.

[2] Die Diphtheritis und der Croup des Rachens und der Luftwege in anatom. Beziehung. Archiv der Heilkunde, VII., p. 481 et seq., 1866.

[3] Das Epithel der Lungenalveolen und seine Bedeutung in der croupösen Pneumonie. Archiv d. Heilkunde, VIII., p. 546 et seq., 1867.

[4] Compare *Bayer*. Ueber die Versuche croupöse Entzündungen der Respirationsorgane künstl. zu erzeugen, u. s. w. Arch. f. Heilkunde, Bd. IX., p. 85 et seq., 1868.

[5] Cellularpathologie, IV. Auflage, p. 476 sqq.

[6] L. c., p. 77.

transudation. In the beginning of every acute inflammation, so long as the number of pus cells mixed with the transuded fluid is not considerable, the extravasated red corpuscles will appear more conspicuous than the white ones. But, besides this, it is to be noticed that the red blood-corpuscles do not possess the power of active locomotion, and therefore, although a few of them may be swept onward by the current of the transudation, the greater number remain in the immediate neighborhood of the capillaries, while the colorless corpuscles migrate to a considerable distance from the vessels from which they have escaped, fill gradually all the meshes of the tissue, and mix with the whole of the exudation. Of not less importance in favor of the colorless corpuscles is the fact that they have a second and very abundant source in the veins. Hence, the more recent the inflammation, the more striking will be the red color of the tissue, surface, and inflammatory product; and, on the other hand, the longer the inflammation continues, the greater will be the excess of the colorless pus-corpuscles, and the more will the color change into gray or yellow. If the inflamed organ, as, for example, the lungs, be supplied with a great abundance of capillaries, the greater will be the predominance of the red color at the commencement of the inflammation."

Furthermore, in croupous pneumonia the coagulated exudation compresses the walls of the capillaries, and thus interferes with the movement of the blood, so as to cause a relative anæmia; *i.e.*, the calibre of the compressed vessels is diminished by the process of coagulation, and but few red blood-corpuscles are found on transverse section. Moreover, changes are gradually developed in the stroma of the lungs; infiltration by fluid and cystoid bodies, and proliferation of the epithelial cells, many of which are found to be multi-nucleated, also take place.

Purulent Infiltration.—The exudation, which has hitherto adhered firmly to the alveolar walls, now separates itself from them; the fibrine, blood, and pus-corpuscles form a uniform granular soft mass, with only here and there remnants of an organic form. These masses often contain lung pigment, and mucine is found to have taken the place of fibrine (Rindfleisch). The exudation disappears partly by fatty degeneration and

absorption, and partly by expectoration, the structure of the lungs remaining intact. The pulmonary epithelium is quickly regenerated, and the vessels become more and more filled with blood. It is a long time, however, before the *restitutio in integrum* is completed, as is shown especially by the deficient elasticity of the affected lung (Rindfleisch).

Rindfleisch is of the opinion that only a small portion of the exudation is absorbed, the greater part being expectorated. This view is opposed to daily clinical experience. It is certainly an exception for large quantities of sputa to be discharged during the reparative stage of a croupous pneumonia, however extensive the inflammation may be. We must therefore conclude that the exudation is removed mainly by absorption. In this process the fatty degeneration plays a more important part than Rindfleisch is willing to ascribe to it, and, on the other hand, the mucous degeneration has less influence than he claims for it. Buhl[1] has also come to the same conclusion as the result of his anatomical investigations, and expresses his opinion concisely as follows: "The whole of the hepatized portion of the lung is restored to its normal condition almost entirely by means of the local absorption of the softened alveolar plugs."

Resolution of an inflammation takes place through the disappearance of the lesion of nutrition in the walls of the vessels, and this is effected by the action of normal blood (Cohnheim). In croupous pneumonia, so long as no further changes take place, the vessels in the inflamed part never become obliterated. They remain pervious, however much compressed by the exudation. This is proved by injecting the lungs from the pulmonary artery; indeed the fact is undisputed. The forces which move the blood remaining intact, there is no reason why the circulation should not be restored.

If stasis take place in the vessels, local death or necrosis ensues in the tissues nourished by them. Cohnheim's views again furnish us with an explanation of this lesion. Gangrene of the lungs is produced in the part which is withdrawn from the influence of the restorative power of the living organism, its decomposition resulting from its exposure to the action of the air and of the parasitic bodies which excite putrefaction; a process similar to the decomposition of albuminous bodies under the same conditions outside of the body. Besides other changes

[1] L. c., p. 30.

there is an evolution of those familiar gases and volatile acids which give to gangrenous fluids such a penetrating and disgusting odor. According to the extent of the local affection we distinguish between circumscribed and diffused gangrene; cavities may be formed in either variety. The sphacelated parts may become capsulated in consequence of the surrounding reactive inflammation. In entire accordance with the theory of Cohnheim is the fact that gangrene of the lungs occurs most frequently where vascular degeneration has previously occurred, as in cachectic individuals, but especially in drunkards. Rindfleisch states that an exudation which contains much blood is apt to produce gangrene. This fact may be explained by Cohnheim's observation, that the number of red corpuscles in an exudation increases with the intensity of the inflammation. It may be mentioned also that gangrene occurs more readily if there be within the infiltrated region a bronchiectatic cavity filled with putrid secretion (Rindfleisch). In this case there is probably a local infection with the bodies which excite putrefaction.

Whenever the suppurative process so far exceeds its usual amount as to break down those elements which form the stroma of the lungs, a *pulmonary abscess* is formed. Generally the large collection of pus is produced by the confluence of several smaller ones. As a rule they become capsulated in consequence of inflammation and proliferation of connective tissue in the surrounding parts. The cavities may partially heal by the process of cicatricial shrinking.

Caseation of the Exudation.—Buhl denies that a caseous pneumonia arising in this way ever develops into phthisis, or that cirrhosis can result from contraction of the parenchyma of the inflamed lung. Clinical experience, it seems to me, proves beyond question that both of these results may occur. So long as it cannot be shown more satisfactorily than Buhl, who is not a practising physician, is able to show it, that the desquamative form of pneumonia, as described by him, cannot be mistaken for croupous pneumonia, the question must remain undecided.

Every physician of much experience knows, that an affection very similar to croupous pneumonia may precede the caseous form of phthisis, or cirrhosis of the

lungs. To be sure, this is as rare as the occurrence of abscesses and gangrene; but, even from Buhl's standpoint, I cannot understand why he should deny the possibility of such an occurrence, especially when he admits "that the purulent infiltration forms a transition to the interstitial varieties of inflammation."[1] On the other hand, it may readily be conceived that, in an organ predisposed to profound lesions, these interstitial changes may be excited by a serious disturbance of nutrition, such as croupous pneumonia unquestionably does induce. Whether the croupous exudation as such can become caseous, we are unable to decide from clinical experience; but Buhl has by no means shown that it is impossible for a "desquamative" to immediately follow a croupous pneumonia, and to run its characteristic course with shrinking, formation of cavities, etc.

The variations in the form of the pneumonia, such as the softness of the infiltration, and smallness of the granules, conditions which give the cut surface a more homogeneous appearance, and which are frequently seen in children, are to be explained by the greater or less amount of fibrine in the exudation, and by the size of the alveoli. These variations are not unfrequently mistaken for other forms of inflammation, especially catarrhal pneumonia. The inflamed lung in croupous pneumonia increases in size, and perhaps becomes even considerably larger than when distended by the deepest inhalation. Of course, the statement of Lebert,[2] that he has seen it enlarged to four or six times its normal size, must be ascribed to a *lapsus memoriæ*, which is not of infrequent occurrence with this prolific writer. In large infiltrations impressions of the ribs are found, especially over the lower parts. Ziemssen asserts that these impressions are caused solely by the violent expirations which occur in severe coughing. I do not see why the stronger resistance which the osseous ribs oppose to the pressure of the enlarging lung is not sufficient to produce a furrow upon it. The intercostal space yields to the pressure, and hence the bulgings, which alternate with the furrows.

Croupous pneumonia attacks some portions of the lungs with greater frequency than others. The annual reports of the Vienna General Hospital, from 1858 to 1870 inclusive, and of the Rudolph Hospital, in the same city, from 1866 to 1869, and for 1871, show that among 7,747 cases the disease affected—

[1] L. c., p. 34.

[2] L. c., p. 659.

The right lung	4,089 times,	= 52.78 per cent.
The left lung	2,902 "	= 37.46 "
Both lungs	756 "	= 9.76 "

With these data let us compare some others, including a large number of cases observed in other places. Grisolle[1] found as a result of his compilations from French, Swiss, and English sources, that among 1,430 cases the disease affected—

The right lung	742 times,	= 51.9 per cent.
The left lung	426 "	= 29.8 "
Both lungs	262 "	= 18.3 "

Magnus Huss,[2] Stockholm Hospital, reports: Among 2,616 cases the disease affected—

The right lung	1,398 times,	= 53 per cent.
The left lung	834 "	= 32 "
Both lungs	384 "	= 15 "

I omit the smaller compilations which have been made by other writers, and which agree essentially with the above, and give the statistics of Ziemssen for pneumonia in childhood.

Among 191 cases the diseases affected—

The right lung	91 times,	= 47.7 per cent.
The left lung	88 "	= 46.0 "
Both lungs	12 "	= 6.3 "

All the observations show that the right lung is more frequently attacked than the left, and that it is affected alone in about half of the whole number of cases. The proportion for the left lung varies between 37 and 30 per cent. in the reports which include adults. I must leave it undecided whether the difference noticed in Ziemssen's statistics for children depends upon the age or is accidental. In the case of double pneumonia in adults, the difference between 10 and 18 per cent., as given in the two tables, is very great; but it should be observed that Grisolle himself regards the figures given by him as too high.

To go more into detail, I have compiled the following table, showing the seat of the pneumonia in 6,666 cases occurring in the Vienna hospitals.

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[1] L. c., p. 42.

[2] L. c., p. 108.

Right lung, 3,580 cases, = 53.7 per cent.			
Upper lobe	810	times, =	12.15 per cent.
Middle lobe	118	" =	1.77 "
Inferior lobe	1,476	" =	22.14 "
Upper and middle lobes	177	" =	2.65 "
Inferior and middle lobes	376	" =	5.64 "
The whole lung	623	" =	9.35 "
Left lung, 2,548 cases, = 38.23 per cent.			
Upper lobe	464	times, =	6.96 per cent.
Inferior lobe	1,515	" =	22.73 "
The whole lung	569	" =	8.54 "
Both lungs, 538 cases, = 8.07 per cent.			
Both upper lobes	73	times, =	1.09 per cent.
Both inferior lobes	223	" =	3.34 "
Right upper and left inferior lobes	43	" } =	1.09 "
Left upper and right inferior lobes	29	" }	
Other combinations	170	" =	2.55 "

Without entering too much into minutiæ, I will merely call attention to the fact, that the inferior lobe was affected at the same time with others, in about three-quarters of all the cases, the right and left sides respectively as seven to six; and that the upper lobe was involved at the same time with others in about two-fifths of all the cases, the right and left sides respectively as five to three.

Various hypotheses have been advanced to explain the greater frequency of pneumonia on the right side. None of them has even a shadow of probability in its favor, and I shall merely refer those who are interested in such details to Grisolle, who has given as an explanation satisfactory to himself, the very obvious fact, that the right lung is larger than the left, and therefore presents a larger surface for the action of the morbific cause.[1]

In rare instances the muscles exhibit the same microscopic appearance which Zenker has so clearly demonstrated in cases of typhoid fever.

The pathological anatomy of the complications need not be considered here.

Analysis of the Individual Symptoms.

Chronology of the Disease.

One would suppose that in a disease so self-limited as croupous pneumonia, there ought to be no difficulty in ascertaining

[1] L. c., p 40 et seq.

its duration, and yet the reverse is the case. Indeed, the point of time which is selected as indicating the commencement of the disease is differently fixed. Many writers reckon from the rigor, since in the great majority of cases the attack is ushered in by this symptom. This is a good selection, because the chill is one of the first symptoms of fever, and after this date the disease may be regarded as continuing so long as the fever lasts, that is, so long as the thermometer shows an elevation of the bodily temperature above the normal.

In practice the instrument is not often available for determining the commencement of the disease, because the patient will not use it to ascertain his temperature in every disturbance of his health. Much may be accomplished by physicians in this direction by popularizing thermometry. Many families which have come under my professional care have become familiar with the use of the instrument, and have in this way saved themselves much anxiety and me many visits. It is to be hoped that the more extensive employment of thermometry among the educated laity will in many diseases be of great service to science, especially in regard to our knowledge of the early symptoms, which can only rarely be observed in a hospital. Any one who has seen for himself how much care is taken in these measurements by those who are instructed as to the value of this mode of investigation, will admit that this hope is well founded.

So long as the thermometer is not in more common use, we must find some sign of more or less approximate accuracy; our best guide is the chill, if there have been one. Besides this, all marked sudden constitutional disturbances, such as convulsions, vomiting, etc., are valuable indications. Local symptoms, pain in the side, etc., are less important. In reckoning the days, or periods of twenty-four hours, the calendar day on which the patient is taken ill is not to be taken as the complete first day, but the reckoning must include the full period of twenty-four hours from the hour of the attack. Thus, if the patient is taken sick at ten o'clock in the evening of January 1, the first day of the disease will last until nine o'clock sixty minutes P.M. of January 2. This obvious rule has not been generally observed.[1] The pneumonia ends when the temperature becomes normal or falls below this point.

The accuracy with which the defervescence is ascertained will depend upon the frequency of the thermometrical measurements. Difficulties may arise if a sequela or a complication follow so immediately as to influence the temperature. In this case our conclusion as to the time when the fundamental disease terminates will necessarily be more or less arbitrary.

In the absence of other signs, we can form only an approximate opinion from the reduction in the frequency of the pulse, the occurrence of sweating, or the improvement in the patient's feelings.

[1] See *F. v. Niemeyer*, Lehrbuch der speciellen Pathologie und Therapie, Bd. 1, p. 193–4. 8. Auflage.

Critical Days.—The old doctrine of critical days, a pet dogma in the Galenic system, experienced so many changes during the middle ages, that the grain of truth in it has been hidden under a mass of additions and non-essentials. It is fortunate that Traube[1] has brought the question forward for reconsideration at a time when the increasing use of thermometry enables us to come to a definite conclusion. Of course the result turns out to be different from what Traube supposed. He states that "the plunging (sprungartig) descent of the temperature in the course of acute diseases which terminate favorably, takes place, if, as usual, it occur before the fourteenth day, always on the third, fifth, seventh, ninth, or eleventh day of the disease."[2] This statement is plainly untenable, and his subsequently repeated assertion that it is so, is no additional evidence to those who prefer to trust to facts rather than authority.[3]

Day of Disease.	Griesinger.[1]	Lebert.[2]	Thomas.[3]	Tophoff.[4] (Naunyn-Frerichs Klinik.)	Traube.[5]	Wunderlich.[6]	Ziemssen.[7]	Total Numbers.	Percentage.
2	..	..	3	1	..	..	..	4	..
3	..	4	9	3	2	10	9	37	5.0
4	2	13	16	5	..	11	3	50	7.0
5	8	36	20	4	7	14	31	120	16.6
6	8	28	18	14	..	14	5	87	12.1
7	14	54	19	16	8	19	35	165	22.9
8	14	52	10	7	..	4	4	91	12.6
9	6	37	2	14	1	3	9	72	10.
10	5	17	4	3	..	..	..	29	4.0
11	3	23	..	1	..	..	8	35	5.0
12	..	9	..	1	..	..	..	10	..
13	..	7	..	..	..	..	3	10	..
14	..	3	..	1	..	..	..	4	..
15	1	2	..	..	..	..	..	3	..
16	..	2	..	1	..	..	..	3	..
17	..	..	..	..	..	..	..	....	..
18	..	1	..	..	..	..	..	1	..
19	..	..	..	..	..	..	..	....	..
						Total, 721 cases.			

SOURCES.

[1] Arch. d. Heilkunde, I., 1860, p. 469.

[2] Klinik der Brustkrankheiten, Bd. I., p. 550.

[3] Archiv der Heilkunde, V., 1864, p. 38. Ibid., VI., 1865, p. 126.

[4] De diebus decretoriis cum exemplis ex pneumonia. Dissert. Berolinens; 1864.

[5] L. c., p. 247.

[6] Handbuch der Pathologie und Therapie, III., 2, p. 347.

[7] L. c., p. 174.

N.B.—This table includes only those cases in which the temperatures have been taken.

[1] *a.* Ueber Krisen und kritische Tage. Anfänglich, 1851–2, in der "Deutschen Klinik" publicirt. Gesammelte Abhandlungen, Bd. II., p. 235 et seq.—*b.* Ueber den Einfluss der kritischen Tage auf die Wirkung der antifebrilen Mittel u. s. w.—Vortrag von 1864. Gesammele Abhandlungen, Bd. II., p. 689 et seq.

[2] L. c., p. 255.

[3] L. c., p. 690.

Of all diseases pneumonia is the one best adapted for the investigation of this point. The preceding table gives a summary of some of the observations made by reliable writers:

It will be seen from the above table that of 721 cases 279, = 38.7 per cent., terminated on the even days, and 442, = 61.3 per cent., on the odd days. The numbers are sufficiently large to refute forever Traube's assertion that the disease ends exclusively on the odd days.

The same result is shown by Fismer's statistics of the Clinic at Basle,[1] but they are compiled on a different plan, and therefore do not admit of direct comparison with those given above. Out of 166 cases the definitive defervescence terminated 93 times, 56 per cent., on the even, and 73 times, 44 per cent., on the odd days. The increased percentage in favor of the even days is probably due to the mode of reckoning.

In general it may be said that, reckoning from the defervescence, pneumonia ends most frequently (64.2 per cent.) from the fifth to the eighth day. The ratio between the even and the odd days is nearly two to three.

Whether the marked differences shown by Ziemssen's figures, as compared with those of the other authorities, are due to any peculiarity of childhood or to local conditions, is an open question. I wish to call attention also to the fact that in Ziemssen's patients the situations of the pneumonia are different from what they are stated to be by other writers.

Fever.—As a rule, pneumonia is accompanied by fever. I do not wish to deny that in a highly cachectic person, who is attacked with pneumonia, the temperature of the body may possibly remain normal; I can only say that I have never yet seen such a case. In pneumonia the fever is not a symptom of such controlling importance as it is in typhus, for the high temperatures which are fatal are very rarely seen. In most cases the fever does not last sufficiently long to become of itself dangerous to life, yet it is a very important symptom.

1. *Temperature.*

The type of the fever in pneumonia is a very simple one;

[1] Die Resultate der Kaltwasserbehandlung bei der acuten croupösen Pneumonie im Baseler Spitale. Deutsches Arch. f. klin. Medicin, Bd. XI., 1873, p. 391 et seq.

within a few hours the temperature rises rapidly as high as from three and a half to five and a half degrees above the normal, continues at this point with morning remissions and evening exacerbations, which average from two to three and a half degrees, and on the occurrence of defervescence falls to the normal, this reduction generally occupying a few hours, sometimes days. The reduction may be interrupted by temporary elevations.

Considered as a whole, the fever in pneumonia is a continued one; that is, excluding the periods of elevation and depression, the fever curve is made up of the normal curve + an increased temperature of *a* degrees. This increase of temperature remains constant; the normal curve shows the regular daily alternations, in which variations may occur within certain limits on different days. This view, which is the one accepted by Liebermeister[1] for the curve of typhoid fever, avoids unnecessary details, and does not attempt to give the natural history of fever curves. I shall not stop to consider it at further length. For the proof I refer to my work on the subject.[2]

The observations on the temperature during the forming stage of croupous pneumonia are scanty. It is certain, however, that a considerable elevation of temperature takes place within the first few hours.

Ziemssen[3] found in children	Fahr.
Once—Four hours after the initial vomiting	102.5°.
Twice—Twelve hours after the access	103.2° and 104.3°.
Thomas[4] found in adults	
Once—Nine hours after the access	104.9°.
Once—Twenty-three hours after the access	105°.

I am able to give some more examples from my private and polyclinical practice.

1. A girl, three years of age. Taken ill between 7 and 8 o'clock in the evening with a chill and severe vomiting. At 11 o'clock P.M., and three or four hours later, temperature 105.8°. Measurements, repeated every two hours, except during the night, averaged 104.5°.

2. Boy, nine years old. Taken ill about 1 o'clock P.M. with severe cerebral

[1] See pp. 78–80, Vol. I., of this Cyclopædia.

[2] Die Körperwärme des gesunden Menschen. Leipzig, F. C. W. Vogel, 1873.

[3] L. c., p. 202.

[4] Arch. der Heilkunde, V., 1864, p. 31.

symptoms. First measurement about 5 P.M.—also four hours later—104.7°. Subsequent measurements, notwithstanding a bath of two hours, always over 104°.

8. Woman, fifty-six years old. Taken ill about noon with severe rigor. First measurement 8 o'clock P.M., 104.3°. Subsequent measurements showed high fever, diminishing towards morning, but were of no value on account of the administration of thirty grains of quinine, part of which was vomited.

The observations of the temperature towards the end of the first, and at the beginning of the second day, are more numerous. They show that the average temperature at these periods is about 104°. Thomas uses the thermometer in the axilla; Ziemssen and myself in the rectum.

At the height of the disease the temperature ranges, as a rule, from 104° to 105°. Extreme elevations may last for some time or only temporarily. We must distinguish here between pro-agonistic temperatures and such as precede convalescence. Wunderlich[1] has observed pro-agonistic temperatures as high as 109.2° (axilla). In cases which have terminated favorably Thomas has observed 106.5°, Lebert 107.2° (axilla), and I also 107.2° on the seventh day of a pneumonia complicated by delirium tremens.

The locality of the disease has some influence upon the temperature. The fever is generally higher in pneumonia of the upper lobe than in pneumonia of the lower. The temperature varies also with the age of the patient. The ranges are generally higher before puberty than afterwards. In regard to individual conditions our information is not very reliable. I wish to call attention to one fact which I have noticed. Women who are attacked with pneumonia just before the menstrual epoch usually have very high temperatures, which continue until the catamenia appear, and then, as soon as only a few drops of blood are discharged, subside considerably. The temperature is usually low in previously cachectic persons, and also in the aged.

Deviations from the regular course of the daily curve are not uncommon. Sometimes the mean temperature increases from day to day, and the exacerbations become longer, the remissions shorter; but generally the reverse is the case; after the second or third day the temperature falls, but only a few tenths of a degree. Intercurrent downfalls are not infrequent; such a reduction often takes place the day before the crisis, but immediately before the beginning of the defervescence the temperature rises again to a considerable height (perturbatio critica).

[1] Das Verhalten der Eigenwärme in Krankheiten, p. 358.

The correctness of all these propositions, for which we are indebted to Wunderlich and his followers, is established beyond doubt.

As far as the *quality* of the fever in pneumonia is concerned, it must be regarded as of a very favorable character. The time which elapses between the use of agents which reduce the temperature, and the fall of the latter to its former height, may serve as a standard for estimating the quality of a fever. In pneumonia the effects of quinine and cold water show that in the great majority of cases the fever opposes only a slight resistance to treatment; *i.e.*, within a given time fewer baths are necessary, in order to maintain the temperature below 104°, than are required, for example, in a typhus fever in the second week. Quinine generally acts more powerfully.

Accurate figures for comparison are difficult to obtain; the above statements are rather a general impression derived from large experience. I will, however, mention that in the Basle hospital[1] it was found necessary in cases of pneumonia to use on an average only fourteen baths of a temperature of 68° and ten minutes' duration in order to maintain the axillary temperature below 102.2°. Sixty-four per cent. of the cases were admitted on or before the fourth day. Antipyretics were used in addition to the baths.

To this slight intensity of the pneumonic fever may be referred the individuality which is so strongly marked in this disease. The same thing is seen in the milder forms of typhoid fever. In all febrile conditions of a favorable nature there seems to be an effort to keep as near the normal temperature as possible, and this tendency is especially noticeable in pneumonia if the patient's constitution be a strong one. So also variations occur in the course of the daily temperature, similar to those which some investigations of mine have shown to exist in the healthy individual. The tendency to intercurrent downfalls, upon which Wunderlich lays much stress, and which are almost characteristic of pneumonia, may be explained by the relatively slight pertinacity of the factor which elevates the temperature.

Besides the continued type of fever, there are also relapsing and intermitting forms.

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[1] *Fismer*, l. c.

The *stage of defervescence* of the fever usually begins late in the evening, a time of day when the normal curve shows a tendency to fall to a lower temperature.

Those writers who have made any mention of their observations on this point are unanimous in regard to the preference of the time of day mentioned. "The defervescence in the majority of cases begins late in the evening, sometimes as early as the afternoon, or in others during the night, comparatively seldom in the morning or at noon" (Wunderlich). "The beginning of the downfall takes place usually late in the evening, more rarely in the afternoon. I have never yet seen the beginning in the morning" (Lebert). "When the temperature fell rapidly, the decline almost always began in the evening or late in the afternoon" (Fismer).

Usually it takes about sixteen hours for the temperature to fall to the normal; but sometimes more, and at others less time is required.

When for therapeutic purposes the measurements are taken more frequently, say every two hours, and a large number of such observations are collected, this question will be settled more clearly than it has been hitherto. I have made accurate records of only a few cases of very rapid defervescence. Once, where measurements were made every five minutes, it took five hours and twenty minutes for the temperature to fall from 104° to 100°.[1]

I have seen one case of a child, in whom the defervescence occupied only four hours. Instances of six hours are even more frequent. Fismer's experience confirms this statement. Lebert gives the following table for the duration of the critical downfall:

12 hours	45 cases.
24 "	25 "
36 "	40 "
48 "	12 "
60 "	2 "
72 "	1 case.

Wunderlich remarks that in most cases the fall to the normal is completed within from twenty-four to thirty-six hours. On the intervening evening the sinking either continues more slowly or is interrupted by a more or less considerable rise.

As a rule, when the downfall occupies more than thirty-six hours we do not regard it as a termination by crisis. There has been much dispute in regard to the distinction between a crisis and a lysis, but certainly the crisis ought not to be allowed to include more than seventy-two hours. There is danger, moreover, that

[1] See Curve 12, in my "Klinische Studien über die Behandlung des Abdominaltyphus mittelst des kalten Wassers." Leipzig, 1866, F. C. W. Vogel.

actual sequelæ, relapses, or recrudescences may be concealed under this convenient term, and may not be distinguished from each other. This is in every respect undesirable, and must introduce confusion into the theory of croupous pneumonia. In my opinion, when the fever has lasted more than fourteen days without the occurrence of a relapse or a recrudescence, and without resolution of the local symptoms, we should be very suspicious that we are dealing with something else than a croupous pneumonia. Either there has been no croupous pneumonia, but rather the desquamative form of the disease described by Buhl, or else there is superadded to the croupous pneumonia a more or less serious destruction of the lung resulting from the antecedent disease. In either case, even if the patient recover after the lapse of weeks, there is no propriety in describing the case as one of slow resolution of a croupous pneumonia. A similar caution is necessary in the case of other complications, such as meningitis, parotitis, etc. This is self-evident, but what is the record upon this point?

I select a work, which must inspire confidence from the place where it is published. In the bulletin of the Vienna Academy may be found some communications from L. v. Schrötter on pneumonia crouposa. In the first of these he publishes the results of twelve autopsies.[1]

In only six of them was the croupous pneumonia uncomplicated. In three of the six other cases there was also purulent meningitis, and in the three remaining, other forms of pulmonary diseases; at least in two of the latter lesions were found, which can only be attributed to pulmonary phthisis, yet no distinction is made in his report, and it is stated that in one of the cases (Kabal) the temperature fell on the eighteenth day, and in another (Prikil) on the twenty-fifth. This entire lack of discrimination makes the communications worthless, and it is for this reason that I have not criticised them more in detail.

It very often happens, especially when the temperature falls rapidly, that immediately after the defervescence the thermometer sinks even below the normal. This occurs less frequently when the defervescence is protracted. With this low temperature the normal daily variations may continue; indeed this is the rule. The whole curve is merely lowered from 1.8° to 2.7°. This continues from one to three days. The lowest absolute temperature which I have observed, was 96.8° (rectum). Similar statements are made by other writers. Perhaps it may be allowable to regard this symptom, which is observed also after intermittent and recurrent attacks, as a "compensation" in the sense explained by me elsewhere.[2]

[1] L. c, Mathemat.-naturwissenschaftliche Classe, Bd. 58, II. Abtheilung, p. 333 et seq., 1868.

[2] Compare Die Körperwärme des gesunden Menschen, p. 59.

Symptoms of the Circulation.

In croupous pneumonia the condition of the circulation deserves the most careful attention. So far as I know, sphygmographic examinations have not been made upon any extensive scale. It is desirable that this deficiency should be remedied as soon as possible. For the present we must be content with the results obtained by merely feeling the pulse. This ancient method of physical diagnosis has been somewhat neglected since the development of the scientific method. In our desire to express all values in figures, we have been satisfied with estimating the *frequency* of the pulse, to the neglect of its *quality*. In pneumonia the careful examination of the character of each pulsation is indispensable.

The pulse in this affection, as a rule, increases in rapidity from the beginning of the invasion, and this acceleration continues at least so long as the temperature remains above the normal. When defervescence occurs, the pulse generally falls with the temperature, and not infrequently, during this period, becomes considerably slower than the normal frequency. It may be reduced to thirty or forty beats per minute. Occasionally, however, especially where there has been previous feeble action of the heart, the pulse becomes very rapid when the reduction of temperature takes place.

In general, it may be said that in a previously healthy adult, when the temperature averages 104°, and the pneumonia is at its height, the pulse will be 100. The daily variation of the pulse generally corresponds to the temperature curve; at all events the ranges are higher in the evening than in the morning. Of course cases differ very much in these respects. One of the most important factors is the time of life. In children the pulse is, as is well known, more frequent; up to the age of puberty it is never less than from 120 to 130, usually considerably higher; in nursing children as high as 200.[1] At a more advanced stage it is usually less frequent. The lowest rate which I have observed was 30, with a temperature of 102.2°. Another important factor

[1] More accurate details are given by *v. Ziemssen*, l. c., pp. 217, 218.

is the greater or less irritability of the cardiac muscles, and also of the nerves and muscles of the arteries; its influence is, however, less than one would perhaps, *a priori*, suppose it to be. For, as experience shows, a woman whose temperament is so sensitive that every noise induces palpitation, does not necessarily have an unusually high pulse during pneumonia. Everything which, either permanently or transiently, impairs the functional activity of the muscles of the heart, tends to increase the frequency of the pulse. A rapid pulse consequently shows that such an impairment has taken place. A long continuance of this condition is injurious. This is an acknowledged fact, and yet, in my opinion, its importance has been somewhat exaggerated.

Griesinger's[1] inference, from a small number of cases (seventy-two), that one-third of the patients with a pulse of 120 and upwards die, may be true for the cases of pneumonia treated at the Tübingen clinic, but will hardly admit of universal application. Marked insufficiency of the heart is, of course, quite common in the cases of pneumonia treated here; but these figures seem to show that cardiac disease, with and without valvular affections, must be relatively very large in Tübingen and its neighborhood. In Holstein I have very often seen as rapid a pulse without its proving to be an unfavorable indication.

I do not wish to be misunderstood as at all undervaluing the importance of such excessive frequency of the heart's action, but merely to point out the folly of generalizing too much in regard to it.

Significant as is a rapid pulse, it seems to me that intermissions, or even mere inequalities in the filling of the arteries, are still more indicative of dangerous failure of the heart's action. At first it is noticed that the individual pulsations produce a variable filling of the artery with blood; the beats as felt by the finger differ in force, and the pulse becomes more compressible; then occur waves, which can no longer be appreciated by the touch; in fact, true intermissions. At the same time the pulse increases in frequency, but not necessarily. This state of things may occur at any time during the whole course of the disease,

[1] L. c., p. 471.

but at the time of the defervescence it is so constant a symptom that it may be regarded as the most valuable of all indications of the resolution of the disease. Of course it requires a certain amount of practice and caution to avoid overlooking these slighter disturbances.

At the outset of the disease the pulse is full and hard. In robust persons this condition continues during the height of the disease until the beginning of the defervescence. But in all the cases which do not run a regular course, the quality of the pulse changes temporarily, even at the very outset. It becomes softer, smaller, empty, and unless this condition can be otherwise explained, it indicates a rapid extension of the local affection. This change is announced by the pulse much earlier and more certainly than by all other methods of examination.

Dicrotism, which is undoubtedly an exaggeration of a normal condition, occurs more frequently towards the end of the disease. The detection of this condition will depend very much upon the degree to which the sense of touch has been cultivated. Pathologically the symptom is of no importance.

Although functional derangements of the *heart* are very commonly observed, the more serious lesions are rare. The statements of earlier writers (Piorry, Corvisart), that the cavities frequently become dilated, seem to be based upon the presence of increased area of cardiac dulness, rather than upon post-mortem examination. It is indisputable that in some cases of pneumonia the area of cardiac dulness is enlarged, but the inference drawn from this fact is incorrect. In the great majority of cases there is an evident retraction of the lungs in consequence of the feeble respiration. The lungs approximate to their elastic statical position, and leave the heart less covered than when the respirations are normal. This is proved by the fact that the cardiac dulness diminishes in extent when the lungs are expanded to their former extent by a deep inspiration.[1] Moreover, the accentuation of the diastolic pulmonary sound, a sign which is often

[1] For some additional considerations in regard to the importance of this retraction of the lungs, and in regard to the mistakes in diagnosis, which are made in consequence of overlooking it, see the dissertation written under my direction by Theodor Look, "Die Vergrösserung der Herzdämpfung bei Chlorotischen." Kiel, 1868.

heard, finds a natural explanation in the improved conditions of conduction brought about by the retraction of the lung. The sound is heard most clearly over that part of the pulmonary artery which lies very near the wall of the chest. A stronger action of one-half of the heart can hardly occur without a previous hypertrophy of the muscles on that side. Undulations or pulsations in the jugular veins are rarely seen. The condensation of the portion of the lung overlying the heart may render the movements of the latter against the chest-wall more forcible, but this is not a constant occurrence.[1]

In the course of pneumonia *functional derangements* of the heart are often noticed, at least for a time. These derangements may be of all grades, from slight irregularities in the order of the pulsations, or inequalities in their force, up to a rapidly fatal paralysis. It is unnecessary to reproduce here Wunderlich's classic description of the collapse.[2] He distinguishes, and very justly, between two forms of collapse, one with increased, the other with diminished temperature of the trunk. Either of these may occur in pneumonia, and we should be careful not to confound the one with the other.

At the very outset of the disease symptoms of exhaustion of the heart may arise, either when the invasion is violent, with a temperature of 105.8°, while the local lesions are comparatively slight, as, for instance, in pneumonia of the apex; or when, as in double pneumonia, a large portion of the lungs is rapidly rendered useless by solid infiltrations. In the latter case a high fever is unnecessary for the occurrence of collapse; but in both instances the collapse is accompanied by increased temperature of the trunk. During the course of the disease failure of the heart's action takes place whenever the local process progresses extensively within a short time, but the temperature need not thereby be essentially changed. The lesser degrees of this condition are very often met with when a careful examination is made for them. I have already called attention to the character of the pulse in this state of things. If the infiltration of the lung is extensive, it is not uncommon for attacks of syncope to

[1] See *Grisolle*, l. c., pp. 254, 255.

[2] Arch. der Heilkunde, Bd. II., 1861, p. 289.

occur, when the patient is obliged to sit upright for a length of time. This should be borne in mind in making an examination. I have seen alarming weakness of the heart's action follow immediately under such circumstances. The usual causes of collapse, such as loss of blood, severe diarrhœa, etc., may, of course, also contribute to such a result.

Collapse, with lowering of the temperature of the trunk, often occurs at the end of the disease, sometimes also in connection with the intermissions. The more rapidly the temperature falls, the more readily does this collapse ensue. In this variety the pulse usually becomes slow, full, and compressible; whereas in the other forms it is rapid and small. This form of collapse is rarely as dangerous as the other.

In both forms of collapse a functional derangement of the heart is the *conditio sine quâ non*, but the derangement may be produced in different ways. In the collapse with high temperature of the trunk, the insufficiency of the heart is produced either by the great and rapid elevation of temperature in connection with an increased obstruction on the part of the circulation (in the vessels of the surface of the body during the chill, and in the lungs as a consequence of the engorgement), the dominant factor being the increase of heat, as it is in attacks of intermittent fever; or it may be caused by the sudden compression of a large number of efferent vessels (Abzugskanälen) in the lung, a condition similar to what occurs in pneumothorax and in rapidly forming extensive pleuritic effusions. Collapse with a diminished temperature of the trunk results, in my opinion, from a reduction of the temperature. We know that a high temperature acts upon the heart as an irritant.[1] Now, if towards the end of the disease this irritant be suddenly withdrawn, the heart, which during the fever has evidently been injured in its anatomical structure, cannot at once dispense with the abnormal stimulant, and there consequently results an impairment of function corresponding to the abatement of the irritation. This condition we call collapse. The results of remedies, which are used in accordance with this view, are convincing evidence of its truth. That cardiac insufficiency does not occur in every case of pneumonia, depends upon individual peculiarities, and is no more surprising than that every patient who has a temperature of 104° is not delirious.

The more serious lesions of the heart—endocarditis and pericarditis—we shall discuss among the complications. In general, it may be said that the occurrence of collapse is favored by all conditions which depress the heart's action, and by the impoverishment of its muscular substance, resulting from acute or chronic diseases.

[1] See *Liebermeister*, Deutsches Archiv für klin. Medicin, Bd. I., p. 461 et seq., 1865.

The *chill*, which is the initial symptom of many cases of pneumonia, requires further notice. As a rule, there is but a single severe rigor, but this is not always the case. Sometimes there are several marked chills, at others only a slight chilliness. Upon the whole, this symptom occurs in about half the cases of croupous pneumonia, either at the very outset, or at least during the first twelve or twenty-four hours. In small children and feeble persons, and especially in the corpulent, it is less frequent than in robust adults. The severe chill lasts but a few hours, four or six at the most, reckoning from the beginning to the end of the chilly sensations.

The statements of writers in regard to the frequency of the initial chill, are derived mostly from hospital patients, the majority of whom are at a vigorous time of life, "secondary" pneumonia not being included. Lebert states that among his patients of this class, 92.5 per cent. had a rigor. Grisolle, among 182 cases of pneumonia, found that a chill occurred in 145 (= 80 per cent.) within twelve hours from the beginning of the disease, and that in 110 of these the chill was the very first symptom. Louis,[1] in 79 cases, found a chill in 61 during the first twenty-four hours. The most precise statistics are given by Fismer. Among 219 cases of pneumonia there occurred—

A rigor	84 times,	= 38.4	per cent.
Marked chilliness without rigor	41 "	= 19.0	"
A slight chill	42 "	= 19.2	"
Chill expressly denied	35 "	= 16.0	"
Chill not mentioned	17 "	= 7.4	"

Sweating may take place at any time during the course of the disease. During the first few days the skin is generally dry, but even at this time a slight temporary perspiration may be noticed. More profuse sweating, occurring early in connection with a high temperature, incomplete filling of the arteries, and marked cyanosis, belongs to the symptoms of collapse, and is a very unfavorable omen, unless it be premonitory of an unusually early defervescence.

In connection with the intermissions of fever, there is often a moderate perspiration, which is noticed first and most conspicuously on the face. With the beginning of the defervescence, which, as already stated, is first announced by the pulse, the sweating increases. It first appears profusely on the forehead and nose, and afterwards, when the temperature has fallen still lower, it extends to the body. So long

[1] See *Grisolle*, l. c., p. 189.

as the defervescence proper continues, the quantity of the secretion is often considerable. It was called "a warm vaporous sweat" at a time when pathology regarded it as a form of the crisis. Entirely distinct from these sweatings are those which precede the death struggle. In these cases, the skin is cold and cyanotic, and bathed in a profuse, oftentimes clammy "colliquative" perspiration. As the pneumonia resolves, the sweating follows pretty nearly the course of the temperature, and generally ceases when the latter has reached its lowest point. If the defervescence reach its completion by successive steps, the same is the case with the perspiration. The most profuse perspirations are seen in children, the slightest in the aged. The different forms of sudamia (miliaria alba and rubra) also occur in pneumonia, but not nearly so frequently as in typhoid fever.

Although our knowledge of the physiology of the perspiration is meagre, the distention of the capillaries with blood is acknowledged to be one of the causal conditions. We know, also, that this symptom may be produced either by an increased afflux, or by a retarded efflux of blood, and that not only the force of the heart, but also the greater or less resistance in the smaller arteries and veins resulting from their regular compression by muscular contraction, exert an important influence. But farther than this the analysis cannot yet be carried. Some have supposed that the evaporation of the sweat, by withdrawing heat from the body, has much to do with the sudden decline of temperature. Independently of other considerations, it must be borne in mind that as the body is surrounded in bed by a layer of air saturated with aqueous vapor, the conditions are unfavorable for evaporation.

The *respiratory organs*, in which the pneumonic process is localized, naturally exhibit the most severe derangements of function. The respiration is always increased in frequency, and often to a marked degree.

In children, forty to eighty respirations per minute are not uncommon; the younger the child the greater, *cæteris paribus*, is the rapidity. In adults and older children this frequency is unusual. On the average it ranges from thirty to forty; I have never seen it above fifty-six, except just before death, or in complications. Grisolle states that in one case, which recovered, it was as high as seventy-five to eighty, and Wunderlich mentions a similar instance. Lebert speaks of sixty-four to sixty-eight respirations; Grisolle gives the minimum as twenty.

I ascribe far less importance to the absolute frequency than to the fact that in croupous pneumonia the *pulse-respiration ratio is always different from the normal.* This condition is of greater significance than that of either of the two functions considered separately. In health, the ratio is about $\frac{2}{9}$, one respiration to four and a half pulsations. In fevers, which run

their course without serious lesions of the lungs or heart, the above ratio remains unaltered, although the numbers expressing the ratio are greater. In croupous pneumonia the respirations tend to become proportionately more rapid than the pulse. This is noticeable even before anything can be detected by a physical examination, and continues after the decline of the temperature. Diagnostically as well as prognostically this symptom is therefore of the utmost importance. In severe cases the frequency of the respiration approaches that of the pulse; the former may equal, and even exceed the latter. Such cases, however, are very rare, but I have met with them, especially when the arteries were in a high degree atheromatous.

It is unnecessary to dwell particularly on the fact that the alteration of the pulse-respiration ratio is not to be regarded as a pathognomonic sign of croupous pneumonia. This symptom always occurs whenever there is fever in connection with a rapid diminution of the respiratory surface. But in croupous pneumonia the functional disturbance often precedes the local lesion, and persists even after its disappearance; hence the clinical value of the symptom. Below will be found some cases in illustration.

Male, nineteen years of age. Taken ill on February 13, 1872, at 3 P.M., with a chill. Examined February 14, 2.30 P.M. Physical exploration negative. Temperature 105.8°, pulse 129, respiration 46. $\frac{P}{R} = \frac{129}{46} = \frac{2.8}{1}$. A pneumonia of upper lobe of the right lung developed itself and lasted seven days. On the seventh day the temperature was 102.5°, and the most marked alteration of the ratio $\frac{P}{R} = \frac{93}{54} = \frac{1.7}{1}$.

Male, sixty-four years of age. Taken ill January 22, 1874, at 8 P.M., with a chill. Pneumonia of inferior lobe of left lung. Duration of the fever, five days. At the end of the first stage of the disease T. = 102.7°. $\frac{P}{R} = \frac{100}{36} = \frac{2.8}{1}$. On the nineteenth day T. = 99.3°. $\frac{P}{R} = \frac{54}{24} = \frac{2.3}{1}$. On the twenty-fourth day T. = 99.3°. $\frac{P}{R} = \frac{58}{16} = \frac{3.6}{1}$. After this date condition normal. In this case the inflammation, which was very superficial, presented local signs during all this time.

The disproportion between the frequency of the pulse and that of the respiration may perhaps be explained by the diminution in the excretion of carbonic acid from the blood. The excess of carbonic acid thus retained would stimulate the spinal cord, and through it the district of the vagus, in such a way that

the respirations would be increased and the cardiac contractions diminished in frequency. This explanation would be admissible if it could be shown that there really was an excess of carbonic acid in the blood in pneumonia.

The effect of cold baths and remissions of temperature is to render the pulse-respiration ratio much more natural.

This is shown by a case published by Traube.[1]

Pleuropneumonia dextra. Third day of the disease, $\frac{P}{R} = \frac{96}{56} = \frac{1.7}{1}$. A hip-bath of 77° F. and thirty-eight minutes' duration. Sixty-four minutes afterwards $\frac{P}{R} = \frac{84}{22} = \frac{3.8}{1}$.

Whether we explain the effect of the bath by an increased excretion or by a diminished formation of carbonic acid, in either case the amount of the poison in the blood is lessened after the bath, and the explanation above given would be confirmed by treatment.

In croupous pneumonia the type of the respiration is not invariably abnormal. The only change noticed may be that the accessory muscles of respiration of the first order (the sterno-cleido-mastoids, the upper part of the trapezii, the scaleni, etc.) are called into play, and this always occurs whenever the breathing is hurried.

If there be much pain, the patient tries to restrain the movements of the ribs by lying upon the affected side, by the use of a bandage or firm pressure, or by bending the chest considerably to one side so that the ribs are brought nearer together. I have no doubt that the slight movement of the affected side during inspiration may often be attributed to a slight and scarcely noticeable bending of the spinal column.

Cough is generally a very early symptom, making its appearance within the first few hours after the commencement of the disease. The paroxysms are of short duration, and are interrupted before they are fully completed; the inspirations which precede them are superficial and are cut short as much as possible.

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[1] Gesammelte Abhandlungen, II., pp. 94, 95.

Up to the height of the disease the character of each paroxysm is the same, whatever its duration. The hardness of the cough first begins to disappear when the defervescence sets in, but sometimes not until later. The cough usually occurs in paroxysms, which last for several minutes. An energetic action of the will, interposed at the very beginning, will often so far suppress the development of the attack that there will be only a single cough, or a few short ones. The frequency of the cough varies considerably in different individuals. No satisfactory explanation has been given for this fact. In the pneumonia of the aged the cough is sometimes entirely absent, or there is only now and then a scarcely noticeable attempt at a paroxysm. This is the case also in the pneumonia of cachectic individuals, or when there is mental derangement such as delirium. If the absence of cough were due to a diminished excitability of the medulla oblongata, in consequence of defective supply of oxygen, we should have an explanation of the cessation of cough, which occurs when the heart has become much exhausted. But this theory will obviously not apply to the frequent absence of cough in otherwise very hearty persons when they have merely reached a more advanced age. In croupous pneumonia the cough is rarely of any service; it is always troublesome and frequently dangerous.[1] It is unusual for the bronchial secretion to accumulate so as to obstruct the air-passages; this occurs only where there is an extensive catarrh (which should then be regarded rather as a complication), or an œdema of the lungs, in which the cough is of no avail. Some good might be accomplished if the paroxysms of coughing were energetic enough to expand the partially collapsed parts of the lungs, but they are too feeble for this purpose. The cough is troublesome because it is painful and interrupts the sleep. In many cases it is excited by every attempt to swallow, and rather than submit to the distress which this act occasions, the patient prefers to endure the annoying thirst, and will sometimes reject every medicine which is offered to him by the mouth. This is most apt to occur when the patient is made half-unconscious by the carbonic acid narco-

[1] I have seen one case of a child, two years of age, where, through the violence of the cough, the ribs became dislocated forwards upon the sternum.

sis, at the very time when there is most occasion for active treatment. The cough may in this way become a source of actual danger to the patient.

The avoidance of deep inspirations, on account of the cough excited by them, also contributes to disturb the aeration of the blood.

The distress of the cough arises chiefly from the *pain* produced by it. This pain originates principally in the inflamed pleura (the lung itself is well known to be nearly insensible); but in addition to this, there is also a considerable amount of irritation of a painful character in the air-passages, even as high up as the epiglottis. Still another cause of pain is the rupturing to a greater or less extent of the respiratory muscles, an accident of not infrequent occurrence. When the cough has been severe the rectus abdominis is sometimes so tender on pressure that we must seek some other explanation than mere muscular fatigue. The intercostal spaces also are often more or less tender beyond the limits of the pleuritic inflammation. This myalgia impedes the respiration, and is, in my opinion, a more important symptom than it has usually been thought to be.

The pain in the side, which is due to the extension of the inflammatory process to the pleura, has from of old been regarded as one of the characteristic symptoms of croupous pneumonia. It generally makes its appearance very early.

Grisolle[1] states that out of 182 patients the pain was felt by 161 within the first twelve hours; of the remaining 21 persons by 17 within the first twenty-four hours, and by only 4 on the second and fourth days.

When the pneumonia begins centrally, and then gradually extends to the surface, the pain may be absent for some time; this is the rule, but there are exceptions. So also in the pneumonia of the apex there is generally no pain, at least in the inflamed part; but later in the disease pain is present, usually, however, in another situation. Nor does the extent of the pain correspond, except in rare cases, to the limits of the pleuritic inflammation, but generally much exceeds it. Whether the pain

[1] L. c., p. 197

radiates along the distribution of the nerves, or whether a slight inflammation extends from the pleura costalis to the muscles, are points yet to be decided. The general opinion favors the hypothesis of a myositis, because the pain produced by pressure upon the wall of the chest is by no means limited to the course of the nerves, but is diffuse, dull, and indefinitely localized, except in the part overlying the inflammation in the pleura. Corresponding to the seat of the pneumonia, the pain is generally felt in the neighborhood of the nipple, and at the lower part of the chest nearly as far back as the posterior axillary line. From these points it may radiate still farther, sometimes apparently following the tracks of the nerves (pain in the upper part of the neighborhood of the shoulder, in the hip, etc.).

In rare instances it is stated to occur on the side which is free from inflammation, while the inflamed side itself is painless. This fact, which has been noticed by good observers (Laënnec, Magnus Huss, etc.), has been unjustly doubted, but I can corroborate it. In explanation of this remarkable symptom Gerhardt has advanced the hypothesis that there are anastomoses between the intercostal nerves of the two sides, and this view seems to have been confirmed by the recent investigations of Magnus Huss.[1] The pain in the side usually subsides towards the end of the disease—frequently after only a few days; but there are numerous exceptions. Persons advanced in age usually suffer but little, and delirious patients scarcely ever make any complaint.

It is not quite clear what is the immediate cause of the pain. Some have ascribed it to a simple rubbing of one pleural surface over the other, others to an inflammation of the neurilemma of the intercostal nerves; but against both of these explanations valid objections have been urged by Wintrich.[2] I prefer the hypothesis that the nerves of the pleura are subjected by the inflammatory swelling of the membrane to a mechanical—perhaps from the nature of the exudation a chemical—irritation, which keeps them continually in a state of such excitability, that slight changes in their position, injuries, etc., are sufficient to produce pain. We find a close parallel to this condition in ordinary whitlow. Strong external pressure, such, for instance, as is produced by lying upon the diseased side for some

[1] Ueber den anderseitigen pleuritischen Schmerz. Deutsches Arch. f. klin. Medicin, Bd. 9, pp. 242–245.

[2] L. c., p. 275.

time, serves to prevent the friction. This explanation enables us also to understand the cessation of the pain in the later periods of the inflammation, when both the mechanical and the chemical irritations have lost their intensity, and why local remedies are useful, such as bloodletting, counter-irritants, cold, and narcotics.

In former times much importance was ascribed to the sense of oppression, not amounting to actual pain, which commonly accompanies severe pneumonia, involving a large extent of lung. This feeling of weight or pressure in the chest, which has a more or less marked resemblance to the sensations peculiar to præcordial distress, must be regarded as a general feeling (Allgemeingefühl), although this term is rather indefinite.

The relations of the pain to the cough have already been indirectly mentioned. They may be described briefly by saying that the cough excites the pain, and the pain the cough. Between these two symptoms a parallelism may almost always be noticed, but of course not an absolute coincidence. Physiologically their intimate connection can be satisfactorily explained by the numerous relations of the vagus to other nerves, and its wide distribution.

In the detailed description already given of the pathological changes in the lungs, the physical conditions were considered, which enable us to recognize the abnormal physical signs in the affected portion of the lung. These abnormal signs depend in brief upon these facts:

1. The lung is filled, not with air, but with a semi-solid tenacious substance, which occupies equally the alveoli and bronchi;

2. The pulmonary tissue, wherever it becomes infiltrated, loses its elasticity;

3. The inflammatory tumefaction of the bronchi produces a narrowing of their calibre beyond the limits of the hepatized parts;

4. The lung tissue adjacent to the hepatized part undergoes a certain amount of compression.

From these preliminary conditions it is easy to draw up a scheme of the physical changes which occur in pneumonia; but to unravel at the bedside the confusion of signs, so as to be sure that there is an infiltration of the lung tissue, is, in many cases, a difficult task. If the hepatization be solid, extensive, and sit-

uated near the surface, the diagnosis will be comparatively easy even for a tyro; but the case is very different when the affected part is small in size and occupies a central position. One method of describing the physical signs is to associate them with the pathological stages to which they correspond. This plan is well adapted for classical cases of the disease, if at the same time special attention be called to the fact that the various changes progress side by side with each other. A better plan, in my opinion, is the one adopted by F. v. Niemeyer in his text-book, viz., to consider each method of physical diagnosis by itself.

Inspection.

A certain amount of cyanosis is almost always present during the whole course of the disease so long as the fever continues. The best place to look for it is around the angles of the mouth, as far up as the nose. On these parts the yellowish-white color presents a strong contrast with the slightly bluish-red tint seen on the cheeks and lips. Usually the face is not uniformly congested; the forehead is more frequently of a pale yellow color, and only the parts mentioned appear injected. The alæ nasi move, but sometimes only so slightly that it requires close observation to detect the motion; the symptom is scarcely ever absent. The acceleration of the breathing is always evident to the experienced eye, and the same is true of the tension of the sterno-cleido-mastoid muscles and the upper parts of the trapezius. In the examination of the chest the difference in motion on the two sides is not so easily discovered. If the infiltration be extensive, and especially if the respiration be very painful, one notices a slight curvature of the spinal column, causing an approximation of the ribs of the affected side, and consequently an imperfect expansion on inspiration in that half of the chest. When asked to take a deeper breath, the patient either complies hesitatingly, or refuses on account of the pain he says it causes him. The character of the cough shows clearly that the paroxysms are interrupted by the voluntary action of the patient. The only exception to this rule is in the case of delirious patients, who will often in a boastful manner distend the chest with air,

until checked by the cough excited by the deep inspiration. I have never observed in uncomplicated pneumonia the girdle-shaped sinking in of the lower part of the thorax. Until I have evidence to the contrary, I shall regard this symptom as always indicative of an extensive catarrh in the lower portions of the lungs. A detailed description of the carbonic-acid poisoning, which is now and then seen in the course of this disease, belongs more appropriately to the article on catarrhal pneumonia.

Mensuration.

The measurement of the circumference of the chest is increased on the affected side when the infiltration is extensive, especially when it is situated in the inferior and anterior part of the lung.

Wintrich,[1] who is my authority upon this point, advises that the comparison of the two sides be made after strong expiration. He has found the difference to be from ½ to 2½ ctm. In all comparative measurements of the chest it should be borne in mind that the side corresponding to the arm generally used is larger than the other; but the difference in the case of the right-armed is greater (½–2 ctm.) than in the left-armed (¼–¾ ctm.). Ziemssen,[2] who has shown that the same fact is true for childhood, states that he has found the average difference up to five years of age to be 1–1½ ctm., the maximum 2 ctm.

On account of the great annoyance which the patient unavoidably suffers when this method of examination is used with due care, it must always play a subordinate part in diagnosis.

Palpation.

I wish here to make a preliminary remark in regard to the "palpatory percussion" of Wintrich.[3] This mode of examination is of the greatest importance in the pneumonia of children. It depends, as is well known, upon the greater or less sense of resistance which the thorax communicates to the sense of touch. The education of this sense is usually much neglected in comparison with that of hearing. To quote a characteristic saying

[1] L. c., pp. 84, 85.
[2] L. c., pp. 236, 237.
[3] L. c., p. 62 et seq.

of Stromeyer, not only the surgeon, but also the physician who treats internal diseases, should "hear with his hands." I have nothing to add to what Wintrich has written upon this subject.

Palpatory percussion shows a distinct increase of resistance at all points where the hepatized lung tissue lies in contact with the walls of the chest.

The examination of the vocal fremitus in croupous pneumonia gives us important information. The statement that in this disease the fremitus is always increased over the infiltrated region is altogether too general and dogmatic. The increase can be discovered only when the infiltration is considerable in amount, when all of the hepatized part lies in contact with the chest wall over a sufficiently large space, and when the bronchi extending to it are open. If the hepatized portion be small and surrounded by tissue containing air, the examination for the vocal fremitus must be conducted in a different way from the usual one, if it is to be of any diagnostic value.

When the flat of the hand is pressed strongly upon the chest, it is only occasionally that any information can be gained by the sense of touch. In the more difficult cases a safe opinion can be formed only by following the rule long ago suggested by Wintrich, viz., the gentle contact of the smallest possible surface with the smallest possible surface (the inner side of the hand, the handle of the percussion hammer, a splinter of wood, etc.). This precaution is especially necessary in practice among children, where the examination is more difficult, because we have to deal with small surfaces, and it is in just these cases that mistakes are most apt to be made.

Many writers, and Grisolle especially, state that the vocal fremitus is more frequently diminished than increased over the infiltrated part. This may be the case if the examination be made without proper precautions; but if the patient be made to cough forcibly previous to each examination of the fremitus, and then symmetrical surfaces of not too small a size be carefully compared, it will be found that when repeated examinations are made, some of them will show on the diseased side an increased force in the waves of sound transmitted from the larynx. A single certain detection of increased vocal fremitus is of greater diagnostic value than several failures to obtain it, because wher-

ever it is observed it may be safely inferred that there is a condensation of the lung.

The reason why we so often fail for a time to find an increase of the vocal fremitus is chiefly because the conducting bronchi are filled with secretion. Not only the bronchi of the inflamed section of lung, but also the tubes of the tissue still containing air, in the neighborhood of the hepatized spot, are in a catarrhal condition. The phenomenon which we recognize at a particular part of the surface of the chest as pectoral fremitus, is made up partly of vibrations which have passed through the portions of the lung still containing air, and partly of those which are transmitted through the infiltrated parenchyma. Now, if of 100 bronchial tubes fifty conduct to air-containing and fifty to condensed lung tissue, then an obstruction of the first fifty will in all probability produce a diminution in the pectoral fremitus, although the conditions of conduction are more favorable than usual in the other fifty. In other words, in every croupous pneumonia the effect of the infiltration is to facilitate for the hepatized part the transmission of the waves of sound produced by the vibration of the vocal cords, while the effect of the accompanying catarrh is to impede the transmission for both the hepatized and non-hepatized portions. The strength of the vibrations over the hepatized region depends upon the preponderance of one or the other of these factors. The unfavorable factor, the catarrh, is variable, and its existence can be discovered; hence the probability that at some time during a prolonged examination we shall be able to detect an increase in the fremitus. It is evident from these facts that the smallest possible surfaces on the chest should be examined and compared at a time. If $\frac{A}{B}$ A be not infiltrated, and B infiltrated, while the conducting bronchi of each are affected with catarrh, then in an examination of the whole surface, A B, the conditions for the conduction of the laryngeal vibrations will be more favorable in the hepatized B, more unfavorable in the conducting bronchi of A and B, and indifferent in A. Hence it is far better to examine B alone, because here we have only the more unfavorable conditions of conduction of the bronchi belonging to this portion. It is evident also from the foregoing considerations that similar differences in the vocal fremitus must occur when the pneumonia affects the central part of the lung.

Another point of practical importance is the fact that normally the fremitus is stronger upon the right side than upon the left. Seitz correctly ascribes this to the anatomical conditions, the difference in direction and size between the right and left bronchi. And the fact is the more striking because the muscles on the right side of the chest are usually more developed, and would naturally diminish the force of the fremitus.

Experience shows that at every period of the disease, from

the engorgement to the gray hepatization, the pectoral fremitus may be increased.

In addition to the improved conduction resulting from the fact that the conducting tube, the bronchus, is surrounded by infiltrated lung tissue, we must remember also that the chest wall vibrates somewhat more easily in those places where the diminution of the negative tension of the air within the lung has made the internal and external resistances almost equal (atmospheric pressure).

Percussion.

Percussion over the inflamed portion of the lung which still contains air, but is less elastic than normal on account of the inflammatory process, produces a tympanitic sound, which is unaltered in its pitch by opening or shutting the mouth. This tone is caused mainly by the lung tissue, which is still everywhere capable of vibrating freely (Wintrich). This condition, that is, lung tissue which contains air and is less elastic than usual, is found at the beginning and at the end of pneumonia, and it is at these two periods of the disease that the tympanitic sound is most frequently heard, but with greater clearness at the end than at the beginning. But even at the height of the disease it is seldom absent in the neighborhood of the solidified portion. The anatomical conditions upon which its production depends are quite numerous; the presence of inflammatory œdema, the compression of the surrounding tissue by the infiltration, the retraction of the lung tissue in general on account of the superficial character of the breathing, each of these factors contributes to the effect, but the latter is unable of itself to produce any considerable change in the quality of the sound.

The tympanitic note which is heard in croupous pneumonia is scarcely ever perfectly clear, and is very different from the sound over the retracted but not yet compressed lung in pleuritic effusion. The note in pneumonia is not only much duller, but it is mingled with sounds which can be distinctly perceived even by an imperfectly cultivated musical ear. This is of course due to the fact that the degree of inflammation or the loss of elasticity is not the same in all the parts vibrating together.

As the air disappears from the infiltrated tissue the note

becomes less intense, and finally resembles the sound obtained by percussing the thigh, but not quite so toneless; as Thomas[1] very justly remarks, "it always has a somewhat tympanitic quality." In this case there is another controlling element in the production of the sound besides the elastic membrane; I refer to the columns of air which are inclosed by the bronchi in the infiltrated lung. This can sometimes be clearly demonstrated by the change in pitch which the tympanitic note undergoes when the mouth is opened or shut. This variety of tympanitic sound can usually be found in dense hepatizations of the upper lobes, more readily on the anterior than on the posterior surface of the chest, and more clearly in pneumonia of the left than in that of the right side. A stronger and shorter percussion stroke will often produce a cracked-pot sound. The columns of air which cause this metallic clang, and the interruptions of it, resembling the clinking of coin, are situated in the larger bronchi of the infiltrated section, in the trachea, and cavities of the throat, mouth, and nose (Williams's tracheal tone). Bäumler, to whom we are indebted for a very readable article on this subject,[2] thinks that Williams's tracheal tone cannot occur in infiltrations of the lower lobe. This is contrary to my experience, which is based upon two cases, one of which recovered and the other proved fatal.

A few words in regard to the latter case, in which the evidence was conclusive. It was one of pneumonia of the whole lower lobe of the left lung, occurring during the course of the secondary fever in a patient with small-pox, in the middle of his twentieth year. When the plessimeter was placed at the level of the sixth to seventh ribs, exactly midway between the spinal column and the posterior axillary line, and was struck with a strong and short blow, an exquisitely tympanitic note was heard, which quite unmistakably changed its note of vibration whenever the mouth was opened or closed. We suspected a cavity from pathological causes, such as abscess or gangrene, and were astonished when the autopsy revealed no other lesion than a solid infiltration in the stage of yellow hepatization. The case was observed by Bartels and myself in the medical clinic of Kiel. Afterwards I saw another similar case in the Kiel polyclinic, and showed it to my assistants. It was a pneumonia of the right inferior lobe, which ran throughout a normal course.

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[1] Arch. der Heilkunde, Bd. VII., Jahrg. 1866, p. 93.

[2] Deutsches Arch. für klin. Medicin, Bd. I., p. 145 et seq.

I am very willing to admit that these cases are rare, for out of the very large number of cases of pneumonia seen by me during many years, and carefully examined in regard to this point, the above are the only instances in which the evidence was entirely clear.

Another reason why a perfectly pure tympanitic note, unless it be traceable to Williams's tracheal tone, cannot usually be obtained in croupous pneumonia may be, that in the different parts of the lung percussed at the same time the sound is controlled sometimes by the elastic membrane, and sometimes by the columns of air inclosed in the bronchi, and that the waves thus produced belong to different systems, and have different rates of vibration.

The percussion sound over an infiltrated lung in pneumonia is less intense, chiefly because the lung contains less air. The air, which is the sounding medium thrown into vibrations by the percussion, is therefore more restricted in space. Moreover, there are still other factors, whose relations are less clearly understood; for instance, the increased resistance of the infiltrated tissue requiring for the production of vibrations the application of greater force.

A few words more in regard to the technics of percussion in the examination of croupous inflammation of the lung, and in regard to the more frequent sources of error.

The most common mistake is percussing too forcibly. When the chest wall vibrates easily, as in the case of children, young persons who are not fleshy, and lean old persons, and the infiltrated part to be examined is small in extent, forcible percussion upon a broad plessimeter will frequently produce such strong vibrations in the surrounding parts that the more delicate differences in sound will not be detected. From frequent observation of medical students, who have just been through a course of instruction in percussion, I am satisfied that their habit of trying to display their skill in the art by percussing as loudly as possible arises from their having been accustomed to percuss over condensations of very considerable size. Infiltrations of the tongue-shaped process give rise, when the percussion is too forcible, to the most singular mistakes, on account of the metallic

clang communicated by the stomach and intestine. And these blunders are committed by others besides novices in the art. Mistakes also frequently arise from seeking for the condensation close to the spinal column. When the infiltration is of large size, alterations in the percussion sound may be found there, but not at the beginning of the pneumonia, at least not in the great majority of cases. I am inclined to think that it is most common for the pneumonia of the inferior lobe to appear first in the posterior axillary line near the lower border of the scapula, and that we must look for it at this point in the beginning of the disease.

In children the smaller foci may often be detected more readily by making use of a procedure which is a little different from the usual methods, viz., by percussing around the chest in zones from the front to the back. For this purpose the child should lie upon the lap of its mother, with the arm of the side to be examined removed a little way from the chest, or the child may be allowed to sit up. In the latter case I allow it to put its arms about the mother's neck. Whichever plan is adopted, the head of the child must be supported if we wish to prevent the child's crying. I lay some stress upon this rule, because it is too often neglected. Indeed the slightest reflection will show that it is wrong to require from a dyspnoic child for the fixation of the head a considerable expenditure of muscular force, which ought to be utilized for the respiration.

My experience has convinced me that for one who has not had much experience it is easier to detect the smaller foci by percussing the chest in zones, than by the usual method of comparing with each other symmetrical parts on the two sides.

When the usual plan is followed, satisfactory results in examining children can be obtained only by percussing in immediate succession with the body in at least two positions. If this plan be not adopted the sources of error are very numerous, the examination will have to be repeated, and the diagnosis in many cases will fail to be made. Yet even the most experienced auscultator may be disappointed if the pneumonia have not yet reached the surface of the lung.

Which method is preferable, percussion by the finger or by

the hammer? Each has its advantages. Any one who has to examine in succession five or six muscular young peasants, as not unfrequently happens in country practice, will soon find that there are limits to the skill of even the strongest finger, especially if the physician be burdened with winter clothing and have to carry obstructions by storm before he can get at his patient. In Holstein I have often had to climb over chests, and rummage through thick feather bed-covers in order to reach my patient, who was coiled up in a ball at the farthest end of a triple bedstead, and clad in several woollen jackets to protect him from cold. In such cases the hammer must be used with no feeble hand, if we wish to penetrate so much fat and flesh. On the other hand, if we use such powerful blows in the case of a slender woman lying upon a spring-bed, or in the case of a child, we percuss not only the patient but the bed also, and can hardly expect to obtain a tympanitic or metallic clang. I have seen some gross mistakes made in diagnosis from this cause alone. Of course, gentle percussion can be made even with the hammer, but the novice might better rely upon his finger.

Ziemssen[1] has very properly laid much stress upon the fact that the cries, importunities, and screams of the child produce important changes by altering the physical conditions. He insists, furthermore, that one side of the chest during expiration should not be compared with the other during inspiration.

In regard to the size of the hepatized portion of the lung necessary for detection by percussion, I agree entirely with the statement of Wintrich:[2] "Infiltrated lung tissue, which is devoid of air, and superficially situated, renders the sound shorter and weaker, only when the airless portion is about two inches in diameter, and at least three-quarters of an inch in depth, and then only on gentle percussion." Central infiltrations, with the single exception of those situated at the apex, must be of much larger size in order to be discovered. In this case no accurate rule as to the necessary size can be given.

Auscultation.

Among the auscultatory signs the crepitant râle ranks first.

[1] L. c., p. 22 et seq.

[2] L. c., p. 48.

This sound is so characteristic that when once clearly heard it can easily be recognized again. It must be admitted that the discovery of the crepitant râle by Laënnec has materially contributed to the diagnosis of croupous pneumonia, but he went too far when he asserted that "the crepitant râle is pathognomonic of an inflammatory engorgement of the lung." Since Wintrich's unanswerable demonstration of the conditions under which the crepitant râle occurs, Laënnec's statement is, *a priori*, to be rejected. If the only necessary conditions be that the (perhaps slightly) swollen walls of the bronchi and alveoli should come in contact during expiration, and should be forcibly separated by the inspiration, and if, moreover, a particularly viscid mucus be not indispensable for the production of this adhesion, then there are many conditions which can give rise to crepitant râles. Keeping in mind the two characteristic factors, that the râle is to be heard only during inspiration, and that it has a special quality of sound, like that produced by rubbing a lock of hair near the ear (Williams), or by salt crackling under a gentle heat, we shall find that the râle may occur not only in croupous pneumonia, but also under the following circumstances:

1. In the beginning of œdema of the lungs.

2. In febrile patients with prolonged dorsal decubitus the râle may be heard in the posterior and inferior portions of the lungs, and on the right side more than on the left. The less frequently the patient changes his position, and the more superficial his breathing, the more continuous and the louder the râles. If he be made to sit upright, and then immediately take a deep inspiration, the râles will almost always be heard. This fact is comparatively little known. I was myself unaware that Walshe, Barth and Roger, and Wintrich[1] have already mentioned it.

Acute rheumatism of the joints, typhus, and surgical injuries, which cause pain when the position of the body is changed, are the conditions which most frequently give rise to strong crepitation in the portions of the lungs mentioned. In the case of very chlorotic patients, so long as they are not bedridden, these râles are seldom heard.

[1] L. c., p. 167.

3. Crepitant râles are sometimes, though rarely, heard in cases of pleuritic exudation. I have found them when this lesion was clearly unaccompanied by pneumonia, so long as the lung was merely retracted, but not compressed by a moderate amount of effusion. They generally disappeared after a few days.

4. At the outset of acute catarrh of the smaller bronchi crepitation is now and then to be heard ; but here also the sign is rare, and usually of brief duration.

It is evident from the above facts that the crepitant râle is not pathognomonic of croupous pneumonia ; but it is also just as certain that in this disease the râle is almost never absent. The patient should be made to inspire deeply, or, still better, to cough, while he is being auscultated. It is only by the strict observance of this rule that an opinion can be formed in regard to the presence or absence of the crepitant râle.[1] Since it is necessary for the production of the sound that the walls of the bronchi and alveoli should be in contact, and then should be separated by a current of air flowing in during inspiration, it is obvious that crepitation can no longer take place when the walls are kept apart by larger quantities of exudation. Hence, over the parts which have become solid with infiltration, this sign is absent. But it by no means follows from this fact that at the height of the disease no crepitant râle at all can be heard, as many writers have erroneously stated. On the contrary, careful examination will almost always show at this time the presence of crepitation in the neighborhood of the hepatized part. Even in central inflammation of the lungs the râle may often be detected ; but Laënnec makes too sweeping a statement when he says that a comparatively unpractised ear can discover by the crepitation the existence of central infiltrations no larger than an almond ! During the resolution of the pneumonia the crepitant râles are heard over the previously solidified part very loud, much louder than at the beginning of the condensation. These râles have been called the *crepitatio redux*, as distinguished from the *crepitatio indux*. The *crepitatio redux* may outlast for a long time the other local, and also the general symptoms. In

[1] See *Wintrich*, l. c., p. 169.

a man, sixty-five years of age, with moderately emphysematous lungs, I heard marked râles even eighteen days after the defervescence. They were so loud that my assistants were able by means of them to discover the original seat of the pneumonia. In this case there were no sequelæ.

Before the pneumonia has reached the surface of the lung, the breathing which is heard over the part affected is generally different from that heard over the other portions, but the difference is not always of the same character. Now it is sharply defined and puerile, now weak, but still clearly vesicular; at other times mingled with some fine, or coarse, but not sibilant, crackling râles. The signs vary in character, and after a few hours a second examination usually gives very different results. This is very often the case, especially, in practice among children.

The causes which produce these different effects are tolerably well defined. The loud murmur depends upon the rapid breathing, in consequence of which the air rushes in with great velocity. On the other hand, some of the alveoli are prevented from expanding quickly on account of the obstruction in the communicating bronchi, whose mucous membrane is commencing to swell from the catarrhal inflammation. The breathing is, therefore, weaker or stronger, according as one or the other factor predominates for the time being.

In the further development of the local lesion we find that along with the increasing number of the crackling râles of different clang-tints (Klangfarbe),[1] there is shortly noticed a distinct prolongation of the expiratory sound. Then the crepitant râles are heard, the vesicular murmur grows more and more indistinct, and is replaced by a blowing sound, and the crackling râles become sibilant. Finally, when the hepatization becomes complete, and the consolidated spot lies immediately in contact with the chest-wall, we hear loud, blowing respiration and strong bronchophony. Sometimes both of these sounds are so intense as to be painful to the ear. When resolution takes place these signs recur, but the râles are generally louder.

The intensity of the râles will of course depend chiefly upon the openness or

[1] Klangfarbe corresponds to the French timbre. The expression used above is the one adopted by Tyndall in his work on Sound.—TRANSLATOR'S NOTE.

occlusion of the bronchi. If, as is sometimes the case, the patient cannot be induced to take a deep inspiration or to cough, I allow him to count as loudly as possible, not merely for the sake of the bronchophony, but also because this procedure induces more rapid and deeper inspirations. The crying of children does not seriously interfere with the auscultation, after one has had some practice. It is a singular fact that many physicians are unaware that the sense of hearing becomes much more acute if the unemployed ear be closed by the finger. Ziemssen[1] lays considerable stress upon this point. In most cases of examination for pneumonia in adults, it is indifferent whether we use the ear or the stethoscope in auscultation, but in practice among children familiarity with both methods is indispensable. If the ear alone be employed, small pneumonic masses may easily elude detection. On the other hand, much time may be wasted if we do not know how to obtain a certain amount of preliminary information by means of immediate auscultation.—Auscultation very often reveals the existence of pneumonia when other methods fail to detect it. The clearness of the laryngeal or bronchial respiration, which occurs in croupous pneumonia when the infiltration of the lung becomes solidified, depends not only upon the improved conducting power resulting from the consolidation of the tissue surrounding the bronchial tubes, but also upon the disappearance of the vesicular breathing, which generally fails to be heard because it is overpowered by the louder conducted sound.

I wish in this connection to call attention to a phenomenon of frequent occurrence, which may lead to mistakes. Sometimes, with extensive consolidation of one inferior lobe, there will be heard on the fourth or fifth day, seldom earlier, signs which seem to indicate infiltration on the opposite, and hitherto sound side, beginning at the lowest part, and gradually extending upwards. The percussion note is slightly dull and of a tympanitic character, and crepitant râles with blowing respiration and sonorous rhonchi are heard. These signs usually extend only a few centimetres beyond the spinal column, but occasionally as far back as the posterior axillary line, and almost as high as the spine of the scapula. The anatomical condition in the more marked cases is a retraction, together with commencing hypostasis in the otherwise healthy lung.

Only a few weeks ago I had an opportunity of demonstrating this phenomenon to my class at the bedside, and afterwards at the autopsy. The patient was an emaciated old woman, and the signs referred to had consequently been unusually well marked during life, and were heard over a considerable surface. The tympanitic, slightly dull percussion note was evidently due to the retraction of the

[1] L. c., p. 29.

lung and the diminution in the amount of contained air; crepitant râles had also been heard. The bronchial breathing and the sibilant rhonchi had been conducted from the diseased side. These sounds were observed with the more clearness, and were less overpowered, because the vesicular respiratory murmur had become enfeebled in the retracted lung.

In these cases a very careful examination is our only safeguard against mistakes. Attention should be directed to the following points:—

1. The bronchial breathing becomes stronger the nearer we approach the diseased side.

2. The vesicular murmur may be heard through the blowing sound, especially if the patient be made to respire deeply, and then the crepitant râles will often disappear.

3. The constitutional symptoms must also be carefully considered. Double pneumonia generally produces serious derangement in the circulation of the blood, whereas with the signs above described there is no special disturbance of this kind. Cases will occur, however, in which this distinction cannot be relied upon. If one is familiar with the phenomenon, and traces its gradual development, he will generally be able to form a positive opinion.

The signs of *pleuritis* occurring in connection with pneumonia are by no means distinctive. It should be remembered that in pneumonia of the inferior lobe the pleuritic friction sound, if present, is frequently to be heard in the axillary line. The diagnosis of effusion is uncertain unless it be considerable in amount. Traube[1] has remarked that under some circumstances, when the lung is firmly hepatized and completely adherent, the cavity of the chest will appear to be distended by a crescent-shaped medium which weakens the sound, a sign which is usually regarded as certainly indicative of pleuritic effusion. I have myself made the same observation in two cases.

In general terms it may be said that an exudation impairs the conducting power and weakens the sound, while the contrary is the case with a pulmonary infiltration. Hence even with constantly open bronchi, the auscultatory evidence of pleuritic exudation will consist of the decided preponderance of the first of these factors over the other. The occurrence of signs of displacement will, on the other

[1] Gesammelte Abhandlungen, Bd. II., p. 854.

hand, be rather favored than otherwise by the condensation of the lung, but a considerable amount of fluid is necessary before the dislocation of the adjacent organs manifests itself by unmistakable signs.

It sometimes happens that a pneumonia of an upper lobe runs its course accompanied by pleuritic exudation, while the lower lobe remains unaffected. The exudation in this case usually collects in the lower part of the pleuritic cavity, and may be discovered by the usual methods.[1]

In the majority of cases of croupous pneumonia the *expectoration* exhibits characteristic peculiarities. *It contains a large amount of mucine intimately mixed with uniformly diffused red blood-corpuscles, and, after a while, dichotomous fibrinous casts from the smaller bronchi.* These ingredients are, of course, present only in the expectoration which comes directly from the diseased portion of the lung. The bronchi, which are affected with catarrh, produce a secretion like that of ordinary catarrh. Usually the sputa discharged in the beginning of pneumonia are purely catarrhal, then the specific ingredients become mixed with them, and finally disappear, while the catarrhal sputa continue alone. Buhl[2] correctly maintains that most of the characteristic expectoration of pneumonia, *i.e.*, the portion which contains blood and fibrine, comes not from the parenchyma of the lungs, but from the bronchial mucous membrane.

The firmly adhesive and very viscid properties of pneumonic sputa are due to the large amount of *mucine* contained in them. The finely divided state of the air and blood is due to their intimate mixture with the sputa by the concussions of severe cough, while the great resistance of the fluid prevents their easily changing the position thus given to them. The larger quantities of mucus are usually found in the expectoration which is not mixed with blood. A diminution in the viscidity is first noticed towards the end of the disease.

Red blood-corpuscles are suspended in the mucus in variable quantities. The color thus produced is usually designated as "rust-brown," or "brick-red." It is hardly worth while to

[1] See also the section on complications.

[2] Zwölf Briefe, l. c., p. 30.

give a detailed description of the very numerous shades of color produced by the admixture of blood-corpuscles, and by the chemical changes which gradually take place under the action of the air upon the hæmatine of the blood. Those who are interested in the subject will find an exhaustive description given by Grisolle. For a description of the characteristic alterations in the expectoration, which imply changes in the local process, *e.g.*, the black-green sputa of pulmonary gangrene, the grass-green of abscess of the lungs, etc., the reader is referred to the articles on these affections.

Remak was the first to call attention to the fact that casts of the smaller bronchi are to be found in the expectoration of pneumonia during the height of the disease. It is not always easy to demonstrate their presence. The sputum should be shaken in water, and then the suspicious little grayish-white particles, which are best distinguished from the rest of the mass by their color, should be separated from it. Remak supposes that these casts are formed from the fibrine set free by the inflammation. Grisolle, Gubler, and Küss maintain that the fibrine of the blood plays the most important part in their formation. The last two of these writers have found ciliated epithelium still covered with cilia on the coagula. This is their chief reason for opposing the theory of Remak. Besides these formed elements there are also found in the pneumonic sputum, upon microscopic examination, a very large number of *pus-corpuscles*, together with epithelial cells, more or less altered, from the air-passages. The chemical analysis of the expectoration gives results of little value, unless we ascribe importance to the large quantity of mucine and to the trace of iron in the ashes. Whether sugar can or cannot be discovered in the pneumonic sputa by means of the reduction of the oxide of copper, as in Trommer's test, is quite a matter of indifference, because there is no way of separating the expectoration from the secretion of the mouth. It appears from a note in Grisolle that Walshe has ascribed an undue importance to this reaction. If the fluids of the tissues are saturated with *bile pigment*, its presence in the sputa may be detected by the usual reactions.

[1] L. c., p. 212 et seq.

The pneumonic sputa, which contain blood and large quantities of mucine, make their appearance in many cases very early in the disease. Grisolle states that in one hundred and thirty-one patients he observed them on the first day forty-five times, on the second thirty-one times, and on the third and fourth fourteen times each. As a rule this characteristic expectoration continues until the cessation of the constitutional symptoms, but it is more bloody during the first few days; at least it appears to be so, but perhaps the subsequent decomposition of the coloring matter of the blood may account for this difference.

The quantity of the pneumonic expectoration is not very considerable. Riesell-Huppert[1] has carefully weighed the quantities expectorated, and has found during the height of the disease a maximum of 67.3 grammes (1,038.68 grains), with 5 grammes (77.16 grains) of dried residue; and a minimum of 35.7 grammes (550.87 grains), with 1.9 grammes (29.32 grains) of dried residue. In the case reported the whole of the left lung was consolidated. During the period of absorption of the exudation the quantity of expectoration fell finally to 8.8 grammes (135.81 grains), with 1 gramme (15.43 grains) of dried residue.

It is by no means an uncommon occurrence for the *characteristic expectoration to be entirely absent.* Buhl's explanation, that in these cases the alveolar croup extends but a short distance beyond the infundibula, I regard as satisfactory; at least so far as the anatomical conditions are concerned.

Young children generally swallow the sputa, so that it is impossible to estimate the quantity, but it is usually more abundant than in older persons; there are numerous exceptions, however, to this rule. In the pneumonia of cachectic individuals and drunkards, the expectoration, if there be any at all, is usually inconsiderable. Under all these circumstances the sputa may be merely catarrhal, and in the aged this is the rule.

Among the anomalies presented by the expectoration in pneumonia, the first to be considered is the more copious admixture with blood, or, in other words, the occurrence of actual hemorrhage. I think we have no right to deny, without further evi-

[1] Untersuchungen über den Stickstoffumsatz in einem Falle von Pneumonie. Leipziger Dissertation von 1869.

dence, that considerable hemorrhages may occur in croupous pneumonia; but it cannot be too strongly insisted that such an occurrence is extremely rare.

During the past twelve years I have seen at least one thousand cases of pneumonia, and I cannot recall a single uncomplicated case in which anything occurred which could be regarded as a hemorrhage. The danger of confounding croupous pneumonia with Buhl's desquamative pneumonia is very great, and in many cases scarcely to be avoided. In other cases scorbutus, purpura hæmorrhagica, or some other similar affection may coexist with the pneumonia. It is not easy to distinguish these cases at first sight, but on more careful examination they will be found to differ anatomically from the croupous form of the disease; so that it is indispensable, in my opinion, to regard the "pneumonia hæmorrhagica" as a distinct affection. It may be difficult to draw sharp distinctions in such cases, as, for instance, between croupous pneumonia and the pneumonia of drunkards, but the attempt must some day be made.

The presence of a somewhat larger amount of blood than usual in the expectoration is unimportant, and we should be careful not to draw prognostic conclusions from such an occurrence. Some writers maintain that a considerable pulmonary hemorrhage is less ominous if it occur during a croupous pneumonia than if it take place apparently spontaneously.

The "pneumonic sputum" is generally regarded as a pathognomonic symptom. Its viscidity and the finely divided state of the red blood-corpuscles in the expectoration are generally regarded as characteristic. The evidence from this symptom is inconclusive, unless fibrinous casts are also shown to be present. I have several times seen an expectoration, exactly similar in other respects to the pneumonic sputa, in general miliary tuberculosis which was localized especially in the lungs.[1]

If there be any considerable amount of *œdema of the lungs*, it may be recognized by the diminished viscidity of the expectoration, which is also more albuminous and contains larger bubbles of air.

Other anomalies will be considered among the terminations or complications of croupous pneumonia. It may, however, be mentioned here, that as death approaches the expectoration often ceases entirely.

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[1] See my article: "Zur Diagnose der acuten Miliartuberkulose." Berlin. klin. Wochenschrift, 1872, No. 5.

Condition of Nutrition.

In the present condition of our knowledge no other symptom seems to have so important an influence upon the nutritive changes in the body as the fever. If the nature of this influence were to be investigated in a thoroughly scientific manner, it would be necessary to answer the following questions: Do the lesions of the respiratory surface make any difference in the amount of débris which accumulate in the body as the result of the fever? Or does the withdrawal of matter from the body by means of the exudation have any subsequent effect upon the total metamorphosis effected by the fever and the respiratory derangement together? It is only necessary to state these problems to show how far we are from their solution.

In regard to the destructive metamorphosis in fever, we have no satisfactory information. No one accustomed to strictly scientific modes of thought will be at all satisfied by the recent work of Senator.[1] No amount of honest labor can replace the lack of critical acumen which is so evident in these investigations.

I have already referred to the investigations of Riesell and Huppert, which were specially directed to an examination of the nitrogenous changes in croupous pneumonia. Conducted with scientific caution, and distinguishing clearly between hypothesis and facts, they are extraordinarily well adapted to show the difficulties which beset our efforts to acquire information upon these points.

The writers examined by strict methods the nitrogen ingested, and the amount discharged through the urine, fæces, and expectoration in a patient twenty-five years of age ill with pneumonia. The examination began forty hours after the commencement of the disease, and extended over twenty-eight days. This space of time is divided by the writers into separate periods.

A. *High Fever* (5 days).

Nitrogen ingested = 2.95 grm. (45.52 grains.)
" discharged = 86.35 " (1,332.69 grains.)
Weight of body at the beginning of the period = 53.279 kilo. (117.42 lbs.)
" " " end " " = 52.600 " (115.93 lbs.)
Difference in weight of body..... = 0.679 " (1.49 lbs.)

[1] Untersuchungen über den fieberhaften Process und seine Behandlung. Berlin, 1873, Hirschwald.

The excess of nitrogen which was derived from the body was 83.35 grm. (1283.39 grains), which, according to Voit's calculations, corresponds to about 2.45 kilo. (5.39 lbs.) of flesh, or about ten per cent. of the whole amount of the patient's flesh at the time the investigation was begun.

B. *Absorption of the Exudation* (6 days).

Nitrogen ingested = 42.96 grm. (663.02 grains.)
" discharged = 135.44 " (2,090.32 grains.)
Weight of body at the beginning of the period = 52.600 kilo. (115.93 lbs.)
" " " end " " = 51.098 " (112.61 lbs.)
Difference in weight of body..... = 1.502 " (3.32 lbs.)

Excess of nitrogen derived from the body = 92.48 grm. (1,427.30 grains). This somewhat larger loss of body weight should not be regarded as wholly due to destructive metamorphosis of the tissues, because during this period the exudation was being absorbed and its nitrogen was passing into the blood and being burned. In what quantity? (The body weight on the sixth day of the disease was more than 2.5 kilo. (5.5 lbs.) less than it was twenty-four hours before. If this statement is not a typographical error I am at a loss to explain it.) At any rate this much may be said: During the whole period of eleven days the body of the patient consumed much more nitrogen than it received, and this deficit amounted to about one-fifth of the whole quantity of nitrogen in the body at the beginning of the period.

C. *Beginning of Convalescence* (7 days).

Nitrogen ingested = 150.72 grm. (2,326.15 grains.)
" discharged = 164.17 " (2,533.73 grains.)
Excess derived from the body = 13.45 " (207.58 grains.)
Weight of body at the beginning of the period = 51.098 kilo. (112.61 lbs.)
" " " end " " = 50.200 " (110.64 lbs.)
Difference in weight of body..... = 0.898 " (1.97 lbs.)

D. *Restoration and Increase of Nitrogen* (7 days).

Nitrogen ingested = 184.47 grm. (2,847.03 grains.)
" discharged = 181.93 " (2,807.83 grains.)
Increase of nitrogen = 2.54 " (39.20 grains.)
Weight of body at the beginning of the period = 50.200 kilo. (110.64 lbs.)
" " " end " " = 50.970 " (112.33 lbs.)
Difference in weight of body..... = 0.770 " (1.69 lbs.)

After the definitive defervescence, which in this case occurred on the seventh day of observation, there still followed twelve days in which excreted nitrogen was derived from the body. Then for the first time the ingested and discharged nitrogen became equal in amount. The increase of nitrogen retained after this was very insignificant. This very laborious investigation gives us very valuable information;

it shows clearly and convincingly that the disturbances which occur in pneumonia are of a very profound character; and, moreover, that the mere observation of the weight of the body is entirely valueless. The total difference is about 3 kilo. (6.61 lbs.), about six per cent., and the quantity of nitrogen in the body is lessened by from twenty to twenty-five per cent. We thus see how little foundation there is for the conclusion, so often drawn in practice, that the restoration is complete when the body has recovered its original weight.

In regard to the discharge of carbonic acid we know almost nothing, not to speak of our ignorance in respect to its formation; while the changes which take place in the non-nitrogenous organic substances are involved in complete obscurity.

At least I am not at all satisfied that any reliance can be placed upon analyses of the air respired for fifteen minutes at a time once or twice daily. Even where the experiments are made at the same hour of the day, with the patient perfectly quiet in bed, and using the same apparatus, I cannot admit that the results are fairly comparable with each other, even though days separated by only a short interval be chosen for comparison. The time of day is here the only constant quantity, especially when the patient is disturbed by the use of the apparatus, and respires under abnormal conditions. The limits of error in such investigations are absolutely indeterminable, as every one must admit. If any one is satisfied with such a result, *habeat sibi.*

In regard to the metamorphosis of the inorganic matters, our ignorance is just as complete. To all appearance the body in croupous pneumonia loses but little of its fulness, except where the treatment has been improper, and regains its former condition very soon after the termination of the fever. The capacity for work, however, requires more time. This fact is more noticeable in persons who belong to the better classes, and are engaged in intellectual pursuits. I do not think that Goethe, four weeks after a pneumonia, would have been able to write a scene of Faust; a wood-cutter by this time could resume his full work.

Organs of Digestion.

In pneumonia *anorexia* is a constant symptom, as it always is in the febrile state. In severe cases it may be even more marked than in other diseases, on account of the urgent difficulty of respiration which occupies the patient's whole atten-

tion. He has no time to hold his breath, no time to swallow, least of all to masticate. Moreover, the act of swallowing frequently excites distressing and long-continued paroxysms of coughing. In many instances the patient endures the tormenting thirst rather than satisfy it at the expense of cough. This is seen most conspicuously in practice among children.

This state of things is one of grave importance, independently of the diminution in the secretion of the digestive juices incident to the febrile condition. One must witness for himself the dread with which everything is rejected that can excite coughing, before he can fully appreciate the danger of this condition. I have seen cases in which it was necessary to give an injection of morphine in the neighborhood of the larynx in order, by local alleviation of the irritable cough, to induce the patient to take a single tablespoonful of a necessary active remedy.

Vomiting is a frequent initial symptom in children, occurring in about half the cases; sometimes it is repeated several times. In adults it occurs more rarely. Many writers, as, for instance, Magnus Huss, ascribe it to an irritation of the vagus, supposed to be produced by the pulmonary inflammation. Granting the possibility of this explanation, it must be remembered that every febrile attack, which sets in suddenly, is very often initiated by vomiting. This symptom occurs during the course of the disease from the following causes:

1. *From cerebral irritation*, especially in pneumonia of the apex. In these cases the vomiting is always violent, generally explosive, comes on suddenly without premonition, and may last for several days. It is troublesome to the patient, but in other respects is without ominous significance.

2. *From severe convulsive cough.* The tough mucus, which clings to the epiglottis, and sometimes the elongated and swollen uvula act during the paroxysms of coughing as mechanical irritants to the mucous membrane of the throat. This is perhaps the most common variety. It may be easily recognized by its being preceded by coughing and gagging.

3. *From indigestion and medicines.* When the attack of pneumonia is ushered in with the stomach full of food, the irritation may arise from this source, and may last for days, especially in children. The same thing may occur in adults, though

with less frequency. It is hardly necessary for me to refer to the emetic action of the various remedies which are in common use in the treatment of pneumonia.

In older persons vomiting from any cause is exceptional.

In at least three-fourths of all the cases the *bowels are constipated*, several days elapsing before a spontaneous discharge takes place. The stool which then occurs is hard, but otherwise presents no characteristic alteration. Diarrhœa sometimes occurs. There may be three or four painless, not very thin discharges. A profuse diarrhœa is unusual, and rarely requires treatment.

The *tongue* presents all the changes which occur in the febrile condition, from a slight coat to the hard, cracked tongue, covered with dirty, bloody sordes. Throat catarrh, with more or less swelling of the uvula, is not at all uncommon.

The derangements of the *stomach* and *intestines* rarely depend upon any more serious lesion than a slight catarrh.

The *liver* takes no direct share in the pneumonic process. In fatal cases it may be engorged from passive congestion. I have sometimes seen perihepatitis on the diaphragmatic side of the organ. The inflammation had arisen in a pleuritis accompanying the pneumonia, and had passed from the diaphragmatic pleura to the muscle, and thence to the covering of the liver. Icterus catarrhalis is not uncommon; according to some writers it is quite a frequent transient symptom. In regard to its significance, see elsewhere.[1]

In opposition to most writers, I have found the *spleen* generally enlarged in pneumonia, sometimes even to a considerable extent. This fact cannot be explained by malaria retained in the system, as Lebert[2] has endeavored to prove by his experience in Breslau in regard to enlargement of the spleen, for in Kiel there was but little intermittent fever, and still less in Tübingen; and yet in both places, according to my experience, splenic enlargement was more frequently present than absent. The spleen does not exhibit any anatomical peculiarities.

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[1] See Forms and Complications of Pneumonia.

[2] L. c., p. 585.

Kidneys and Urine.

In fatal cases of pneumonia changes in the distribution of the blood are found also in the kidneys. When the fever is at all high, catarrh of the pelvis is generally present, extending to the straight renal tubules, and producing desquamation of the epithelium. The more serious lesions do not belong to pneumonia as such.

At the outset of the disease the urine is lessened in quantity, the diminution during the first few days not being very great, but at the height of the attack amounting to about one-half of the normal quantity. For a strong adult the daily average is about from 800 to 1,000 ccm. (1.4 or 1.7 pints), during the later days of the disease. Sometimes it is much smaller, 400 ccm. (0.7 pint), or less. The restoration of the normal quantity does not coincide with the crisis of the disease. Several days always elapse before this occurs. In some cases during the early part of convalescence considerably more than the normal amount is discharged. The polyuria, which is occasionally noticed as a transient symptom in many febrile diseases, I have never seen in pneumonia. The *specific gravity* of the urine is always increased: it ranges, as a rule, at about 1.025; minimum, about 1.019; maximum, 1.030. Lebert gives it as high as 1.035. The color is always darker than natural, between four and six of Neubauer and Vogel's color scale. On cooling, the urine precipitates a reddish-yellow sediment, consisting chiefly of amorphous acid urate of soda, a few crystals of free uric acid, and epithelium of the urinary passages in a more or less advanced condition of fatty degeneration. Sometimes there may also be found isolated crystals of oxalate of lime. This copious sediment makes so strong an impression upon non-professional persons, that the "thick, red urine" is one of the symptoms most frequently remembered and voluntarily mentioned. The *reaction* of the urine is usually acid. The total quantity of acid is really diminished, but relatively it is considerably increased.[1]

[1] *Neubauer-Vogel*, Anleitung zur Analyse des Harns, Zweite Abtheilung.

The *urea* is, perhaps, without exception, increased in pneumonia. The percentage may rise as high as 5 per cent.; the daily quantity discharged may vary from almost entire absence to 70 grm. (1,080 grains) in vigorous persons (Brattler, J. Vogel).

The *uric acid* is not so much increased at the height of the disease as is commonly supposed. A not considerable excess, however, always makes its appearance in the urine discharged from twenty-four to forty-eight hours after the defervescence (Bartels).

The excretion of *chlorine* decreases rapidly from the very commencement of the disease, and may fall to a minimum. Its diminution is not, however, specifically characteristic of croupous pneumonia, because it is found also in all acute febrile affections (J. Vogel).

The *sulphuric acid* is also generally diminished.

The quantity of *phosphoric acid* is much more variable. Sometimes, even at the height of the disease, the daily excretion is much above the normal (J. Vogel).

The time has gone by when a boilery for urine was regarded as indispensable for a physician's scientific progress, and I dare say we are all grateful for this deliverance.

For, the apparent certainty given to the estimation of the nutritive changes by the easily manipulable methods of chemical analysis, even in the hands of the inexperienced, has led to an immense amount of labor, but to extraordinarily few results of scientific or practical value. How little reliance can be placed upon imposing arrays of figures is well shown by the difficulties experienced in the investigations at Munich, in explaining the crude results of single analyses of urine, which probably at no time represented the total daily discharge. The well-proved facts we do possess are only partially susceptible of a physiological explanation. The scantiness of the water discharged in the urine, a symptom which is more noticeable in pneumonia than in other febrile diseases, is probably due to more than one cause. It may be explained by a change in the composition of the blood, induced by the exudation in the lungs, or by altered pressure in the kidneys, in consequence of deranged action of the heart. Here our knowledge ends. The copious sediment is due to the scantiness of the water in the urine. In regard to the increase of urea, I refer to my previous remarks on the excretion of nitrogen. The fact, ascertained by Bartels, that there is always an excess of uric acid discharged after the defervescence, is explained by him as follows: During the continuance of the fever an unusually large amount of nitrogenous matter becomes oxidized, and gradually passes through the different intermediate steps to the formation of urea. At the moment when the fever ceases the conditions become

more unfavorable for oxidation than they were during the height of the fever. Under these circumstances some of the imperfectly formed products, more particularly the uric acid, which have accumulated in large quantities in the tissues, appear in the urine according to their degree of solubility. It cannot be denied that this hypothesis is very plausible.

Chlorine has played a very important part in its time in the pathology of pneumonia. The diminution of this substance in the urine has been asserted to be a diagnostic and prognostic symptom. The careful investigations of J. Vogel have shown the fallacy of this opinion. The lack of chlorine in the urine in febrile diseases may be explained by the fact that, when the supply of chlorine is much diminished, the body retains whatever chlorine is set free by the consumption of the tissues. Kaupp's experiments have demonstrated the possibility of stuffing (chlormästung) of the human body with chlorine; and I have satisfied myself by an investigation upon this point that even much larger quantities than are stated by this writer may be retained for a long time. The physiological relations of *sulphuric acid* are far less understood. It is possible that it also may be retained, and the same may be said of the *phosphoric acid.* In the latter case, however, the conditions are more complicated, because this acid is supposed to have a double source, one in the albuminous substance of the tissues, and the other in the blood.[1] It is obvious also that the inorganic salts derived from the tissues will be found in diminished quantity in the urine in consequence of their passing into the pneumonic exudation, or on account of anomalies in the perspiration and discharges from the bowels. These changes, in order to be clearly understood, must be studied in connection with the total tissue-metamorphosis.

Abnormal Substances in the Urine.

Small quantities of albumen are very often present in the urine of pneumonia. Albumen would probably be found in every case if examinations were made every day. Quantities which can be weighed are comparatively rarely met with. If the amount be at all considerable, a careful search will always reveal casts. Albuminuria has no prognostic significance in croupous pneumonia, or at least only in the exceptional cases, in which it shows the occurrence of acute nephritis or the previous existence of kidney disease.

It deserves to be mentioned here, that when the urine in pneumonia contains a large percentage of urea, nitric acid may produce a precipitate of nitrate of urea, which, upon superficial examination, may be mistaken for albumen. A similar error may arise, though of course less frequently, when a little nitric acid is poured into

[1] *Ludwig*, Physiologie, II., p. 403.

urine containing much neutral urate of soda. The cloudiness in this case depends upon the acid urate. To avoid mistake, the novice in testing urine for albumen should always follow the method recommended originally by Panum, and afterwards popularized by Hoppe-Seyler: to equal quantities of urine and a saturated solution of sulphate of soda, acetic acid is freely added, and the mixture heated. With this precaution deception is impossible.

When pneumonia is complicated by icterus the bile pigment may be discovered in the urine by its well-known reaction with nitric acid containing some nitrous acid.

Care should be taken not to infer the presence of bile pigment solely from the rapid and marked reaction of the coloring matter of the urine with nitrous acid in urine which is very rich in urea,—a mistake not infrequently made in practice. The *green color*, which is the first sign of the oxidation of the bilirubin, must be demonstrated; if it be absent, the evidence is inconclusive.

No other abnormal substances are known to be present in the urine of pneumonia. It is not impossible, however, since Bartel's observation in regard to the increased excretion of uric acid is undoubtedly correct, that, along with the uric acid, other incompletely oxidized derivatives of albumen may be present after the occurrence of defervescence.

Condition of the Skin.—Eruptions.

Mention has already been made of the properties of the perspiration in pneumonia, and also of the occurrence of the characteristic miliary eruption. The most frequent eruption is herpes facialis, which is present in from two-fifths to one-half of all the cases, including all periods of life, and usually makes its appearance from the second to the fifth day of the disease, but in a few instances even later.[1] As a rule, an eruption of herpes is a favorable prognostic symptom.

Few acute febrile diseases are so frequently accompanied by herpes as croupous pneumonia. Intermittent fever comes next in frequency, with about 30 per cent. (Griesinger). Lebert thinks he has observed local differences of frequency; but for Breslau, where herpes is said to be more common than in Zürich, he gives the unusually small proportion of 13 per cent. Drasche[2] gives 40 per cent. for the cases

[1] *Thomas*, Arch. der Heilkunde, VIII. Jahrg., 1867, p. 478.

[2] In Canstatt's Jahresbericht f. 1860, Bd. III., p. 207.

observed in Vienna, Geissler,[1] 43 per cent. for Leipzig. The latter has adduced strong evidence, at least for the 421 cases carefully studied by him, that herpes has a prognostic significance. To reproduce this evidence entire would oblige me to enter too much into details. I select only a few figures. Out of 182 cases of pneumonia, with herpes, 17 died (9.3 per cent.); out of 239, without herpes, 70 (29.3 per cent.). Of the whole number 159 were over thirty years of age. Of these, 50 had herpes, and 10 (20 per cent.) died; 109 had no herpes, and the mortality was 58 (53.2 per cent.). In a few cases herpes may occur elsewhere than on the face.[2]

At present we are wholly unable to explain why herpes should have a favorable prognostic significance.

Occasionally croupous pneumonia is accompanied by an abundant formation of furuncles. Multiple circumscribed gangrene of the skin is rarely seen, except among the starving proletariat in large cities. I have met with it once in a child with pneumonia, living in a dark and badly ventilated cellar.

Cerebral and Nervous Symptoms.

It is first of all necessary to distinguish those symptoms which are due merely to the elevation of temperature, and which vary from the more trifling disturbances, such as headache, sleeplessness, restlessness, to the most severe, such as unconsciousness, involuntary discharges, and furious delirium. It would be difficult to assign any reason why these symptoms, which are common in all febrile conditions, should be ascribed in pneumonia to any other cause than to the fever. And yet some have distinguished one form of pneumonia as "cerebral," "meningitic," etc., on account of the strong predominance of the cerebral symptoms. It is necessary to inquire, therefore, whether in these cerebral cases it is indispensable to suppose that there is any other cause at work, common to all of them, besides the high temperature from fever, in connection with the special susceptibility of the patient. To this question we must give a decided negative. Besides the fever there are probably always individual conditions, which favor the occurrence of cerebral symptoms. Some of these individual factors are known to

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[1] Arch. der Heilkunde, II. Jahrg., 1861, p. 115 et seq.

[2] *Thomas*, Arch. der Heilkunde, Jahrg. VII., 1856, p. 284.

us; we also know that certain peculiarities of the fever may have the same effect.

The attempt[1] made some years ago to disprove the views advanced by Liebermeister, and adopted by the great majority of unprejudiced observers, in regard to the influence of high temperature in producing nervous symptoms, is so puerile that it is hardly worth while to discuss it. If we wish to estimate the action of agents upon the central nervous system, we must always bear in mind the fundamental fact that the idiosyncrasy of the individual, whether it be acquired or be dependent upon primary disposition, has a most radical influence. In regard to the resistance to alkaloids, alcohol, etc., there are very great differences, as every one knows by daily observation. We might as well deny that alcohol will intoxicate, as reject the evidence that an increase of temperature will produce cerebral symptoms. It would be possible to give very good statistical evidence that alcohol is an inert substance. The workmen in the breweries of Munich, who, as Liebig says, are well known to drink on an average sixteen quarts of beer daily, might be cited as evidence of such an assertion.

The special character of the fever, which favors the occurrence of cerebral symptoms, consists mainly of the very rapid and considerable elevation of the temperature.

When the body is artificially heated by the external application of heat, all the symptoms of cerebral disturbance of the first degree are produced just as in fever. In an experiment of mine with the vapor bath[2] the temperature in the rectum rose within thirty-four minutes to 106.5° F. During the first twenty-seven minutes 104° was reached; an elevation of 3.2° occurred very early, and in the last seven minutes one of 2.5°. During this last period the person complained for the first time of vertigo and headache, and afterwards of loss of power over the limbs, staggering, etc. Bartels[3] had before this made the same observation. These cerebral symptoms disappear quickly on rapid cooling, as in fever, provided they have not continued too long, in which case the material changes have, of course, advanced so far that a long continued withdrawal of heat is necessary to restore the balance.

Experience shows that the most severe cerebral symptoms usually occur in connection with pneumonia of the apex setting in with a rapidly developed fever.

Even Heinze is obliged to admit that the cerebral symptoms are more frequently associated with pneumonia of the upper lobes. Out of one hundred and seventeen

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[1] Ueber das Verhältniss der schwereren Kopfsymptome zur Temperatur bei Pneumonie von Dr. O. O. Heinze. Arch. der Heilkunde, IX., 1868, p. 49 et seq.

[2] Deutsches Arch. für klin. Medicin, Bd. IV., p. 357.

[3] Greifswalder Beiträge, Bd. III.

cases in which "the upper lobes were attacked either alone or in connection with other lobes," forty-seven presented severe "head symptoms" (40.2 per cent.). Out of two hundred cases, in which the lower lobes were alone affected, there were only fifty-one in which the symptoms in question were present (25.5 per cent.). The distinction commonly made in regard to this point is, however, really incorrect, for when a pneumonia extends from a lower or middle to the base of the upper lobe, the temperature rises slowly, and does not always reach an unusual height. The second factor, the rapid and excessive elevation of temperature, is absent, inasmuch as the brain has become accustomed to an increased temperature by the previous basic pneumonia. In Heinze's forty-seven cases of pneumonia of the upper lobes there were only twenty-one in which the upper lobes were affected alone. Liebermeister[1] is entirely correct in rejecting such unprofitable statistics as these. And yet every experienced physician, especially if he have seen much of pneumonia in children, knows that it is just in these cases of pneumonia of the apex that meningeal symptoms and high fever are most apt to occur.

The following *individual* conditions are acknowledged to be predisposing causes of severe cerebral symptoms.

1. *Age.*—In children these derangements are much more common than in adults. In after years they are relatively less frequent, probably because the pneumonia is developed more gradually and with much less fever. If, as happens in rare instances, the attack is ushered in with high fever and temperature, the cerebral symptoms are always present.

Only a few weeks ago I saw a case of this kind, which is always rare. A woman, sixty years of age, was suddenly attacked at night with a rigor, succeeded by vomiting, unconsciousness, profound coma, and involuntary discharges from the bowels. In the morning we examined her condition, and found a temperature of 105° F., bloody sputa, and evident infiltration of the right lower lobe. The disease ran a normal course, and terminated in recovery. The cerebral symptoms disappeared in less than two days after the use of antipyretic measures. Comatose condition, somnolency interrupted by slight wandering, and inability to answer questions quickly, are met with in old persons in all slight febrile attacks, as well as in pneumonia.

Alcoholism.—Delirium not infrequently occurs in the pneumonia of persons addicted to the excessive, sometimes only the comparatively excessive use, of alcoholic stimulants. This delirium may be of a non-typical character throughout its whole

[1] Beobachtungen und Versuche über die Anwendung des kalten Wassers bei fieberhaften Krankheiten von *Liebermeister* und *Hagenbach.* Leipzig, Vogel, 1868, p. 87.

course, or it may present all the symptoms of delirium tremens. The non-typical symptoms will usually disappear when sleep is induced by restoring to the brain at the proper time its accustomed stimulus.

The frequency with which one meets this form of cerebral symptoms will of course depend upon the extent to which dram-drinking prevails in the neighborhood. In Kiel these symptoms were of common occurrence; but every year I saw also about fifteen cases of delirium tremens in its most pronounced form. Here, in Tübingen, in about 1,700 cases, I have so far treated only one patient with delirium tremens, and none at all with this particular form of delirium. In Sweden, the classic abode of delirium tremens (nearly 7 per cent. of all the cases of pneumonia suffer from it), Magnus Huss[1] found this form of cerebral disturbance in drunkards very common. It appears to me doubtful whether Grisolle[2] does not go too far, even for his own circle of observation, when he regards alcoholism as the most frequent cause of delirium in pneumonia

3. *Anæmia and Debilitating Treatment.*—Acute cerebral anæmia, even when it has not gone so far as to directly and of itself produce functional derangements of the brain, at least favors their occurrence. Such an anæmia may be caused not only by direct loss of blood, but also by treatment which diminishes the flow of blood to the brain by weakening the action of the heart and producing collapse. The cerebral disturbances which sometimes occur when the defervescence takes place very rapidly, probably have a similar explanation. Those methods of treatment which tend to maintain the functional activity of the heart operate in the opposite direction.

In my experience these forms of cerebral disturbance, especially delirium, have occurred only in connection with loss of blood in abortion. This result I ascribe, at least partially, to my treatment; for a "delirium of collapse"—and as such these forms should be regarded—can scarcely occur when the amount of blood in the brain and the rapidity of its circulation are normal. These symptoms were of very common occurrence at the time when the rage for venesection prevailed.[3]

4. *Intellectual persons of an easily excitable temperament* are more apt than others to present cerebral symptoms.

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[1] L. c., p. 41.

[2] L. c., p. 374.

[3] Compare *Chr. Lutz*, Zur Lehre vom Delirium des Collapses. Deutsches Archiv f. Klin. Medicin, Bd. V., p. 530.

To mention such a cause is really only to explain x by y when they both occur in the same equation. The fact is, however, of practical importance, because when severe brain symptoms are present, we shall feel easier if they occur in a person predisposed to nervous excitement. Every physician of experience knows, moreover, that persons of strong will can often control the delusions of fever by energetically opposing them.

5. Although the number of my observations upon the following point is not large, I wish to call attention to it because it seems to me to be important. Local affections of the brain, with symptoms which are in the main merely functional, may be produced by the influence of radiated heat upon the head, as seen in true sunstroke. In two cases of pneumonia of the inferior lobes, with severe cerebral symptoms, I noticed that just before the outbreak of the disease—*sit venia verbo*—such an irradiation of the scalp had taken place.

In both cases the cause was the walk taken by the pupils of a school. One of the children had his head uncovered; in his hurry not to be too late his cap had been left at home. The other child had repeatedly gone out under a hot July sun with uncovered head, notwithstanding the warning of his teacher. In both instances, simultaneously with the rigor, the temperature rose to an unusual height, and the symptoms were very similar to those of meningitis.

I should not lay so much stress upon this point, if I had not several times seen severe cerebral symptoms in children during pneumonia continue so long as the patient's head was exposed to the radiated heat of a very hot iron stove, and disappear as soon as the position was changed.

Many writers have regarded it important to settle the period of the disease when the cerebral symptoms are most apt to occur. This, in my opinion, is unnecessary; the symptoms may arise at any time, in a marked degree, even after the defervescence, and it may be left to the physician to ascertain the cause in each particular case. When this is done, the symptoms in the great majority of cases lose their importance. Here, if anywhere, the maxim is applicable: "Qui bene diagnoscit, bene medebitur." In my experience this view leads us to tangible results, and is preferable to the mere generality that "the cerebral derangements in pneumonia are to be regarded as essentially

a toxic or infectious encephalopathy."[1] The complication of pneumonia with cerebro-spinal meningitis will be considered under the head of complications.

Blood and Hemorrhages.

As an act of pious duty to the past we must refer to the *changes in the blood.* "Fibrinous crasis," "hyperinosis,"—these are terms which belong to the history of medicine. They are based upon the increase of fibrine in the blood in pneumonia, a fact which must be regarded as settled, notwithstanding the defective methods of analysis and the blunders of those who used them. Aside from the strong influence of blood-letting in increasing the fibrine, the amount of this substance appears constantly, or at least quite constantly, to become greater as the pneumonia progresses. The statement that the formed constituents of the blood are diminished in proportion as the fibrine is increased, seems to me rather apocryphal.[2]

Copious *hemorrhages* rarely occur in pneumonia. Epistaxis is, however, quite common at the beginning of the disease, and sometimes the menstrual flow is excessive. Hemorrhages from the intestines, kidneys, or bladder, I have never seen in uncomplicated cases. If the patient happened to be pregnant and aborted, the hemorrhages were very apt to be profuse and uncontrollable, with a fatal termination. It may be that this result was due to accidental causes; the number of my observations is small, for in the Vienna Hospital from 1858 to 1870 there occurred only twenty-five cases of abortion in pneumonia, and of these only seven proved fatal.

Complications.—Differences in the Course of the Disease.—Forms of Pneumonia.—Relapses.—Terminations.—Sequelæ.

There is no form of disease with which pneumonia may not be associated, especially when the powers of resistance have

[1] *Lebert*, l. c., p. 590.

[2] Those who are interested in such details will find an excellent résumé in Wunderlich's Handbuch, Bd. I., p. 571.

been enfeebled by previous illness. A complete statement of all the complications would have to embrace the whole of pathology. For this reason we shall include under this head only those complications which stand in direct relation to the local affection, or which give to it a special and unusual character. The table below gives a general summary of the more important complications of pneumonia, and is compiled from the statistics of Magnus Huss for Stockholm, Fismer for Basle, and from the records of the general hospital and Rudolph hospital for Vienna.

Diseases.	Vienna. Total number of patients, 5,738.		Stockholm. Total number of patients, 2,616.		Basle. Total number of patients, 230.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
Pleuritis exsudativa	298	5.2	104	4.0	35	15.3
Bronchitis capillaris acuta	..	..	140	5.4	..	..
Bronchitis chronica	..	..	42	1.6	..	..
Emphysema pulmonum	..	..	26	1.0	20	8.7
Tuberculosis pulmonum	60	1.1	36	1.4	4	1.8
Pericarditis	27	0.5	22	0.9	9	3.9
Endocarditis	11	0.2	4	0 2	2	0.9
Valvular diseases of the heart	32	0.6	23	0.9	2	0.9
Meningitis cerebralis	8	0.1	2	0.1	3	1.3
Icterus	30	0.6	23	0.9	65	28.3
Diarrhœa	..	..	160	6.1	25	11.0
Morbus Brightii	66	1.2	52	2.0	(1)	(0.4)
Rheumarthritis acuta	2	..	22	0.9	1	0.4
Delirium tremens	19	0.3	180	6.9	..	..
Alcoholismus chronicus	..	..	16	0.6	14	6.1

This table shows that there are very marked local differences in regard to complications. In one important point, the complication with alcoholism, Vienna and Stockholm differ considerably, although the other percentages are nearly the same. In Vienna 0.3 per cent. delirium tremens, 6.9 per cent. in Stockholm,—certainly a striking contrast. Again, in pleuritis, pericarditis, icterus, and diarrhœa Basle shows percentages considerably in excess of the other cities, but perhaps because the smallness of the statistics gives wider room for the operation of accidental causes; for if we add the 128 cases which belong to classification B in Fismer, the difference is changed. The percentage of pleuritis becomes 17, pericarditis 3.6, icterus only 20.1, and that of diarrhœa is diminished to an extent which cannot be stated exactly. And yet it must be admitted that the

complications of pneumonia are much more numerous in Basle than in the two other cities.

In general, we may say that unfavorable hospital conditions facilitate the occurrence of complications, particularly the stealthy progress of inflammatory processes.

Every one knows this who is familiar with the course of epidemics in hospitals. After many patients have lain in the same wards for a considerable time, the sequelæ will be found to become more numerous, although the original disease has become less intense and the strictest attention has been paid to cleanliness. Of course these remarks do not apply to Basle, where, I am assured by Liebermeister, the conditions are in this respect favorable.

It is desirable to divide the complications of pneumonia into two groups. The first includes *the affections of the respiratory and circulatory organs occurring in connection with pneumonia.*

The statistics of most places except Basle agree in placing the frequency of *pleurisy with fluid effusion* at about 5 per cent. of all the cases. Sometimes the rate is higher; Grisolle gives 12.6 per cent.; Dinstl,[1] for the hospital in Wieden, a suburb of Vienna (1857–61), as high as 15.8 per cent. The conditions upon which these differences in frequency depend are unknown to us. It is only very rarely that the effusion can be detected during the first few days; usually the diagnosis may be made on the fourth or fifth day. In most cases the effusion is serous, with more or less admixture of fibrine. This form may gradually pass into the purulent variety. An effusion which is entirely purulent from the start, if it ever occur, is certainly extremely rare when there is no other complication. In the adult the quantity necessary for certain diagnosis is at least from eleven to fourteen ounces; in children a less quantity, corresponding to the smaller dimensions of the thorax, but even in these cases I could not always make a positive diagnosis unless the fluid amounted to from four to six ounces. If the quantity be smaller, its existence might be suspected, but nothing more. The pleuritic effusion may be very large, but only in cases where there is no considerable infiltration. *If the lung be consolidated*

[1] Canstatt's Jahresbericht f. 1862, Bd. III., p. 220.

from apex to base by pneumonia, there is no room for a large amount of fluid.

The course of the pneumonia is rarely altered by the effusion, unless the latter is so copious that the case is to be regarded rather as a pleurisy with pneumonia than as a pneumonia with pleurisy. The pneumonic fever usually subsides at the proper time, and the temperature may even become normal for the time being. Generally however, even in respect to the fever, the pleuritis takes the place of the pneumonia, and exerts very little influence upon the regressive changes in the lung, or at most it merely retards them for a time. In cases of moderate effusion complicating pneumonia the physical signs should not be accepted as conclusive unless the same results are obtained after repeated examinations. The cautious observer, who does not claim to be infallible in diagnosis, will follow this rule. In regard to the theory of these signs it is sufficient to refer to the explanation previously given of the physical signs of pneumonia. Dulness on percussion, increased resistance, loss of the pectoral fremitus, and feeble respiration—signs which are characteristic of pleuritis—may be present transitorily also in pneumonia. The only sign which is certain is the displacement of adjacent organs, especially the heart; this never occurs in pneumonia to a perceptible degree. But this condition implies, of course, the existence of a large effusion. As for any refinements in the diagnosis of a pleuritic effusion complicating pneumonia, I have none to offer; I can only recommend repeated examinations, the careful comparison of the results obtained, and especially the search for the signs of displacement. To the character of the dulness I ascribe far less importance, and still less to the increase of pain in breathing when pleuritic effusion takes place; for as soon as sufficient fluid has been effused the pain must disappear. If a positive diagnosis be desirable, puncture may be resorted to. For this purpose I prefer a fine canula of Thiersch's syringe. The wound thus made is so slight as to be entirely free from danger, even if the lung be punctured. I have several times been obliged to thrust the trocar through an adherent lung in order to draw off the fluid behind it, and I have never met with

any bad results. In one case, at the autopsy, a slight extravasation of blood was found at the seat of puncture; the patient was *in extremis* on admission, and died about twenty-four hours after the operation. If the pneumonia have resolved, the physical signs of the pleurisy present an uncomplicated character. *The course of pleurisy after pneumonia is generally more favorable and of shorter duration.*

Huss states that the mortality from pleurisy among his cases of pneumonia was only 0.8 per cent. above the average. This agrees very nearly with my own general impression. In Basle it was different, the mortality among the pleuritic patients being more than double the average (16.5 to 40 per cent). In that city empyema was comparatively frequent, but terminated favorably. Chronic pneumonia, with or without tuberculosis, may likewise follow the pleuro-pneumonia; and, in fact, it is quite possible that the antecedent inflammation of the lungs increases the disposition to this affection.

Pleuritic effusion following pneumonia is usually more dangerous in old than in young persons, and chiefly because its course is more tedious.

Extensive *catarrh of the smaller bronchi* is a very dangerous complication, especially in children and the aged. The pneumonia is often concealed by the marked symptoms of bronchitis, and so are its physical signs. It is a question to be decided by careful anatomical examination in each case, whether the disease is not really an acute catarrhal pneumonia. Many of the so-called "soft infiltrations," which are so commonly found in old persons, are certainly not croupous pneumonia. The course of pneumonia with this complication is far less typical than usual; even when there is no doubt of the existence of a croupous pneumonia, the progress of the case rarely has a cyclical character. The temperatures remit on the critical day, but do not fall to the normal. An "amphibolic period" of uncertain duration and course now intervenes, the regressive changes take place gradually and tardily, the original consolidation can be detected for an unusually long time, and the catarrh continues and extends to new portions of the lung, while in other places it disappears. The patient becomes weaker, his appetite fails, and after a long period of confinement to bed finally succumbs with the symptoms of hectic fever. In many cases the autopsy

reveals only an extensive catarrh in addition to the more or less advanced resolution of the original consolidation. But sometimes there are also found patches which have undergone caseous degeneration, proliferation of the connective tissue in the stroma of the lung, or diffuse pleurisies which have resulted in adhesions.[1] At other times death is produced by acute insufficiency of the heart, which has previously manifested itself by more or less strongly pronounced attacks. A complete restoration of health, with disappearance of all the symptoms, is possible, but this always requires at least several months. The above-described course of the disease belongs especially to middle and old age. Complications with chronic bronchitis and chronic pneumonia, or with tuberculosis of the lungs, present a very similar history. In children the catarrh of the smaller bronchi does not often change very essentially the course of the croupous pneumonia. The best evidence of this is the absolutely small percentage of mortality in children with croupous pneumonia. In practice this form of the disease may often be mistaken for acute catarrhal pneumonia, and the error is not always corrected at the autopsy, if the observer be not familiar with the lesions. At all events, the symptoms of the capillary bronchitis predominate over those of the pneumonia.

In persons with considerable *emphysema* of the lungs an attack of pneumonia is very often attended by marked dyspnœa and frequent attacks of cardiac exhaustion. These symptoms are produced not only by the obstruction of the pulmonary circulation in consequence of the emphysema, and by the bronchial catarrh which is seldom absent, but also by the secondary cardiac changes necessarily attendant upon emphysema. The danger in such cases is very serious. If the result be favorable, the local regressive changes always take place slowly, as might be inferred from the diminution of the absorbing surface in emphysematous lungs.

All complications of pneumonia with *diseases of the heart* are especially dangerous to life ; and generally in proportion to the extent to which the function of the heart is impaired. In *val-*

[1] In regard to the possibility of confounding these cases with the "desquamative pneumonia" of Buhl, see the section on pathology.

vular affections (I use the term as a general one, without reference to the presence or absence of compensation) Huss found the mortality to be 30.4 per cent.; in *acute heart diseases*, including endo- and pericarditis, 57.7 per cent.; the average mortality from pneumonia itself in Stockholm being 10.7 per cent. In Basle the deaths with *acute* heart diseases, including acute degeneration, were 66.6 per cent.; with *chronic* affections, including degeneration, even 77.8 per cent. These facts are ample evidence of the dangerous character of this complication.

This complication is not of very frequent occurrence. The maximum frequency is, perhaps, that observed at Basle, which was 4 per cent. When *pericarditis* occurs as a complication the only symptoms, from the beginning to the end of the disease, which indicate its existence are the functional derangement of the heart, as shown by considerable cyanosis, a somewhat increased frequency of respiration, a diminished filling of the arteries, and especially slight intermissions of the pulse, symptoms which differ from each other only quantitatively. Among the subjective symptoms, when the heart affection is severe, the most prominent are the attacks of precordial pain.—In twelve cases of fatal pericarditis Huss found that in ten no resolution of the pneumonia had taken place, and ascribes the result to this cause. I have never been able to trace it to any other cause than failure of the heart's action.—The existence of pericardial effusion cannot always be readily detected by a physical examination. Small quantities of fluid, coexisting with a pneumonia or pleuritis, may easily be overlooked.—The pericardial effusion may be either sero-fibrinous or purulent. When it contains much fibrine, the heart, after the fluid has been mostly absorbed, often becomes surrounded with a laminated coating several layers in thickness, and the inflammation is found to have extended deeply into the muscular substance. I have repeatedly found this condition in children; the course of the disease, which was always fatal, was protracted and the fever moderate. The cases which recover are generally those with a more serous effusion. If extensive adhesions are found between the heart and the pericardium, I of course infer that the effusion contained a large amount of fibrine. When the effusion is purulent the disease

generally runs a more acute course, but not invariably. Facts do not warrant us in laying down an absolute rule upon this point. Pericarditis may occur within the first few days, or it may be detected only after the defervescence of the pneumonia. In the latter case there will always be more or less doubt in regard to the time when the pericarditis occurred. For, even after the irregularity of the pulse is noticed, days and even weeks sometimes elapse before one can be sure that effusion is present. Pericarditis may arise in two ways: by direct extension of a pleuritic inflammation to the pericardium, when the inflamed pleura is in contact with the pericardium; or, where no such direct transition can be discovered, the complication may proceed from a constitutional anomaly, a diathesis, or some other unknown cause. In the first of these two varieties the tongue-shaped process of the upper lobe of the left lung plays a very important part. In the cases observed by me, in which pericardial effusion occurred, this portion of the lung had generally been implicated in the pneumonia. The second mode of origin is more frequent in drunkards and in persons with renal disease, etc., than in those who had previously enjoyed good health.

For the sake of reaching a wider circle of readers, and because the facts possess therapeutic interest, I give below a short sketch of a hitherto unpublished case of pericarditis following pneumonia, in which I performed an operation. A strong boy, six years of age, was suddenly taken ill on April 7, 1872, with a rigor, high fever, and pain in the side. He was received into the Kiel polyclinic on the 9th. Examination showed the existence of a small pneumonic infiltration in the lower part of the left upper lobe. On the 12th, while the disease was running a mild course, the pulse for the first time became intermittent. Continuous but moderate fever, with very marked prostration. On the 22d (the fifteenth day of the disease) pericardial effusion was recognized, the patient on the day previous having been much worse, and the temperature considerably higher. During the following week the effusion increased, with compression of the left lung, the patient became very cyanotic, and had transient attacks of cardiac insufficiency, which daily became more frequent and alarming. On May 1st (twenty-fourth day of disease) I punctured the pericardium, and, by means of Thiersch's syringe, drew off three ounces of pus. Relief was afforded until May 3d, when, the pericardial effusion having increased considerably, the condition of the patient required a second operation. The patient was narcotized by hydrochloric ether, and a hydrocele trocar was used for the puncture. Six and a

half ounces of thick pus were withdrawn. The canula was left in the pericardial cavity, which was washed out four times every day with a one per cent. solution of chloride of sodium warmed to the temperature of the body. The immediate result was very happy. The patient felt better, and the cardiac attacks ceased. The heart approached the chest wall. After the seventh of May (four days after the operation) severe cerebral symptoms, temperature 106° F., Cheyne-Stokes's symptom, convulsions, and coma. Death on the evening of May 8th. Autopsy, 9 A.M. (Cohnheim): Suppurative pericarditis, and partial adhesion between the two surfaces of the pericardium. The opening of the artificial fistula was situated very nearly over the middle of the right ventricle, and the muscular substance had not been touched by the canula or the point of the trocar. Thrombosis of the vena cava superior above the ventricle, and continuation of the same into the sinus sigmoides sinister and the sinus transversus of the same side. Suppurative meningitis of the convexity and base. No tubercles.

The object of the operation was, first of all, to obviate the danger to life from compression of the heart by the effusion; and then, it was hoped, recovery would ensue by the adhesion of the two surfaces of the pericardium. The first result was attained, and the second, as the autopsy showed, was in process of completion. If recovery take place from suppurative pericarditis, as may happen in very rare instances, it will probably always result in complete adhesion between the heart and the pericardial sac. The operation, as I had perhaps for the first time attempted it in this case in accordance with principles which are approved by our experience in pleurisy, is therefore in every respect justifiable, and the result calls urgently for its repetition.

It is scarcely possible to give a general outline of the modifications produced in the symptoms of pneumonia by the intercurrence of *endocarditis*. The symptoms will depend very much upon the extent of the morbid process, and upon the implication of the muscular structure of the heart. Emboli may form and produce infarctions of the spleen and kidneys; these lesions are very frequently mentioned by writers in Basle.

Valvular insufficiency and degenerations of the substance of the heart act deleteriously in proportion as they interfere with the function of the organ. Pneumonia thus complicated usually runs a protracted course.

The complications belonging to the second group stand in no immediate relation to the local affection; they involve other organs besides those of respiration and circulation.

Meningitis may occur in two forms in connection with pneumonia: as an *infectious disease*, meningitis cerebro-spinalis

epidemica, and as a *simple local meningeal inflammation.* The latter variety is at all events rare. One modification of it has already been mentioned (the case of suppurative pericarditis); at other times the genesis is less clear, and pyæmia or some analogous condition may perhaps be inferred.

When an epidemic of infectious meningitis is prevalent, this complication of pneumonia is not very uncommon.

Immermann and Heller [1] have recently called the attention of physicians to this complication. Out of thirty autopsies in cases of pneumonia they found nine in which meningitis was also present. It was ascertained beyond question that an epidemic of cerebro-spinal meningitis was prevailing at the same time.

It appears certain that the pneumonia is the primary, and the cerebro-spinal meningitis the secondary disease. The croupous pneumonia seems to produce a special disposition to the cerebral affection. The writers above mentioned make the following statement: "In none of our cases were the cardinal symptoms of the existing croupous pneumonia altered by the occurrence of the meningitis; in addition to the symptoms of the former, there were generally only occasional single groups of symptoms, which seemed to be due to meningitis." Judging from the few cases which have come under my own observation, I am prepared to accept this view as the correct one.

In Kiel only a few sporadic cases of cerebro-spinal meningitis occurred, but besides these there were isolated cases complicated with pneumonia. Notwithstanding the closest attention to the symptomatology of these cases, I was unable during life to diagnosticate the complication. In one instance, a man, forty-six years of age, the temperature was taken every two hours. The temperature, to be sure, rose steadily towards the end of the disease to above 105° F., but at the same time signs of consolidation were detected at the apex of the left upper lobe, in addition to the original infiltration of the right lower lobe. Who, under these circumstances, would be willing to diagnosticate a cerebro-spinal meningitis from the occurrence of diffuse cerebral symptoms, however severe they might be, and in this case they were entirely absent? The only symptom of any value was the deficient reaction of the temperature under quinine on the day before death. Seventy-five grains of it were given within eight hours, in thirty and forty-five grain doses, and six baths at 77° F., and of twenty minutes' duration, and yet the temperature, taken every two hours, rose steadily to 105°, 105.4°, and finally 106.9°. The pulse remained constantly about 120, and was full and strong. The patient became unconscious only two hours before death. In the case-book it is stated that "even a short time before

[1] Pneumonie und Meningitis. D. Arch. f. klin. Medicin, Bd. V., p. 1 et seq.

this event no headache, stiffness of the neck, or other symptoms of a central character could be ascertained." The autopsy revealed an extensive suppurative meningitis, together with pneumonia of the right lower and left upper lobes. In another case the course of the disease was very similar.

The result is almost always fatal.

Icterus as a complication of pneumonia may be produced in different ways, and varies also very much in its significance. The frequency with which it is detected will depend very much upon the care taken to ascertain its presence. A slight yellow color of the conjunctivæ and skin is often seen, and if these cases be included, the percentage will be high.

This fact is best shown by the observations made at Basle. During one period, in which but little attention was paid to the presence of icterus, this symptom was noticed in only 5.5 per cent. of the cases; while in another period, when the point was not neglected, the percentage was 28.3.

The milder cases of jaundice are very generally, and probably correctly, explained by venous congestion of the liver, compression of the biliary passages by the distended veins, and the diffusion of bile into the blood. These cases have no clinical significance. In regard to the importance of the more severe forms of icterus in pneumonia, there are wide differences in opinions and experience.

If we confine ourselves to the recent period in which exact methods of observation are used, we find that Traube [1] and Mosler [2] ascribe to this symptom an important influence upon the course of pneumonia; especially the latter writer, who characterizes it as in his experience the most dangerous of complications. Out of fifteen patients he lost eleven, a mortality of 73 per cent. Grisolle makes no direct statement upon this point, but it appears that out of his twenty cases he lost only three.[3] Fismer also had a small mortality, 20 per cent., which is not much above the average. Huss lost only 8.7 per cent., and says expressly, "that this complication does not seem to exert any pernicious influence upon the result of inflammation of the lungs." [4] Lebert takes the same ground, and vigorously opposes the frequently described "bilious pneumonia."

I have no opinion of my own in this matter based upon per-

[1] Gesammelte Abhandlungen, Bd. II., p. 691 et seq.

[2] Deutsches Archiv für klin. Medicin, Bd. X., p. 266 et seq.

[3] See also *Grisolle's* remarks on "Bilious Pneumonia," pp. 387–396.

[4] L. c., p. 138. And yet (p. 52) he speaks of headache and prostration as characteristic of the "gastric and bilious" form.

sonal experience. In the few cases which I have observed, the pneumonia ran its regular course undisturbed by any unusual symptom. It is generally admitted, and has been verified by autopsies, that the anatomical cause of severe jaundice is a mechanical closure of the choledochus. Grisolle is the only one who leaves the matter in doubt. Mosler even admits that the symptoms are merely those of jaundice coexisting with those of pneumonia. Traube is not quite clear upon this point. Among the symptoms of a specially dangerous character the following are mentioned:

1. The condition of the nervous system. From the outset marked stupor, diminished sensibility, tendency to delirium.

2. Tendency to meteorism, a symptom involving great danger to life (Traube).

3. Diminished power on the part of the patient to withstand the effects of bloodletting; strong tendency to collapse.

Leyden[1] has found the biliary acids in the urine of his cases of pneumonia in which this complication occurred. His remarks upon this point are very judicious and pertinent.

"In my opinion the complication with jaundice has an important share in the differences which this affection presents, as opposed to the usual form of croupous pneumonia. We are not called upon to explain in detail every unusual symptom of diseases, in which idiosyncrasy, accidental circumstances, the constitutional condition, etc., play an important part. It is fair to suppose, however, that the admixture of biliary acids with the blood is not an indifferent complication in an affection which is by itself a serious one. At all events the biliary acids are present in the blood, and must more or less seriously threaten the blood and the heart, the maintenance of whose functions is of vital importance in pneumonia."

In any attempt to explain the injurious effect of this complication, Leyden insists, and I agree with him, that we should regard the power of the biliary acids to dissolve the red blood-corpuscles and to paralyze the heart, as a fact of prime importance. The extent to which these results may be carried will depend, of course, upon the amount of bile absorbed. However, we should beware of schematizing too much, and of paying too much attention to the jaundice to the neglect of symptoms elsewhere.

[1] Beiträge zur Pathologie des Ikterus, p. 133 et seq. Berlin, Hirschwald, 1866.

In these cases the temperature certainly plays an important part. It may be very high. In the case published by Leyden [1] it averaged 106°; in Mosler's cases also, although intercurrent falls occurred, it was generally high.

The *epidemic* occurrence of "bilious pneumonia" is mentioned also by recent writers; some state that the disease is most prevalent in summer, others in winter.

We must be cautious in drawing conclusions from the accounts of former times, because our predecessors evidently meant an entirely different thing by the term "bilious" from what we understand by it. Mosler has recently fallen into error on this point, probably because he did not have access to the original source, and formed his opinion from a reference in Schmidt's Jahrbuch. He gives an account of "bilious pneumonia" as observed by Dr. Hauff in Besigheim. The original [2] shows indisputably that Hauff had in view nothing more nor less than the usual course of croupous pneumonia, and that the complication with jaundice, if it occurred at all, was at all events uncommon. How far from clear Dr. Hauff himself was in regard to what, in accordance with Stoll's Reminiscences, he calls "bilious," and how little he deserves the eulogies of Mosler is evident from the following passage. Hauff is controverting another writer, Goeden, who "from deference to a theoretical opinion has given a distorted description of the disease, which he states begins with symptoms of hepatitis, or a status gastricus." He goes on to say: [3] "Also after the development of the chest symptoms, the liver symptoms do not immediately recede; in fact their disappearance is as uncommon as for the icteric color of the skin to be a characteristic symptom of the disease. Nor have I ever seen the icteric, *i. e.*, the white, clay-colored stools observed by Goeden." This language is at least clear. Still less can I approve of Mosler's course of supplementing citations from annual reports with American observations in regard to the occurrence of epidemics of "bilious pneumonia." Such observations will rarely stand criticism.

Upon the whole, facts warrant us in rejecting "bilious pneumonia" as a variety of this disease, and the cases so-called should be regarded merely as pneumonia complicated with a mild or severe degree of icterus. For the bilious pneumonia as understood by Stoll is an illusion, and cannot be grasped as a scientific conception, unless the hæmatogenous jaundice, which is invoked by some as *Deus ex machina*, should prove to be something more than an abstraction.

Pneumonia is a very serious and not rare complication of

[1] L. c., p. 138–39.

[2] Journal der praktischen Heilkunde, herausgegeben von Hufeland und Osann, 1834. X. IV. Stück. Octoberheft. p. 77 et seq.

[3] L. c., pp. 87, 88.

kidney diseases, especially the chronic forms. Huss lost 50 per cent. of his cases. The pneumonia is the final symptom, as in cachectic conditions generally. There is nothing characteristic about it to distinguish it from pneumonia occurring in other forms of cachexia, unless it be the occurrence of uræmic symptoms, which may manifest themselves, especially by a high temperature. Huss lays stress upon the fact that the gray hepatization takes place very rapidly; his experience may probably be explained by the large number of drunkards among his cases.

Rheumarthritis acuta and *intermittent fever* were the most frequent acute complications observed by Huss. Their influence upon the mortality is unimportant.

In regard to the dangerous character of the pneumonia of drunkards there is only one opinion. Huss had a comparatively small percentage of deaths, only a little above 20 per cent. Fismer, in Basle, lost, on the other hand, 55 per cent. I have already mentioned the wide differences in the frequency of this complication in different localities.

Huss distinguishes between two very distinct varieties of pneumonia in drunkards. I have nothing to add to his descriptions, as my own experience agrees very nearly with his. He says:

"If an immoderate brandy drinker be attacked with inflammation of the lungs, the seizure generally takes place in the midst of apparently perfect health. In only a few instances have I observed previous symptoms, which usually consisted of gastric derangement, a feeling of faintness, enervation, and loss of sleep. A chill, often a severe one, announces the outbreak of the inflammation. After this event two different conditions may arise; in the one the patient is active in his movements, his face full and eyes glistening; in the other the expression of the face is stupid, flabby, indifferent, and sunken, and the patient feels profoundly prostrated. In the first variety the arms and hands soon begin to tremble, the patient talks incessantly, but usually stammers, delusions occur, at first temporarily but soon continuously, until on the second, third, or fourth day—seldom later—delirium tremens becomes fully developed. Sometimes,

however, this delirium tremens sets in almost immediately after the chill. The delirium lasts from three to five days, seldom longer, and is followed by a condition of more or less profound exhaustion. During the delirium the patient is rarely conscious of any pain in the side, the cough is either absent or insignificant, and usually there is no opportunity to examine the expectoration, so that the diagnosis of pulmonary inflammation must be based exclusively upon the results of percussion and auscultation.

In the second variety there is very early developed an adynamic condition, very similar to that of the "nervous" or "adynamic" form of pneumonia. To these symptoms may also be added the following: trembling of the hands and arms, disconnected stammering speech, either complete insomnia or continual drowsiness, and delusions of a more quiet character, in which the patient mutters to himself, grasps at the air, and picks around him. The tongue, which is very often dry, trembles when protruded, and once extended often remains in that position until the patient is bidden to draw it in again. The first of the two varieties mentioned occurs usually in drunkards who are not so weakened but that they still retain a considerable degree of reactive power; the second, generally in those who are already much enervated, and more or less cachectic." [1] This description, taken from Huss, refers to the completely developed conditions, but in some cases the symptoms assume a milder form. The rigor may be absent, or at least we may be unable to positively ascertain its occurrence. The patient remains out of bed, at least during the first few days; makes himself conspicuous by his great officiousness, and is continually chattering in a confused and nonsensical manner. He obeys every impulse, but the emotion is only momentary, and soon disappears to be replaced by another. In times of political excitement I have seen many cases of this kind, useful tools in the hands of agitators for the purpose of tearing down government proclamations, but good for nothing else notwithstanding all their shouting. As soon as they take to bed, delirium breaks out in full violence, and in the fatal cases a purulent pneumonia is found, although the

[1] L. c., pp. 55, 56.

patient had been on his feet the very day previous. The temperature may be very high; I have seen it at 107.6° F. A further characteristic of the disease is the very great tendency to sudden collapses, which in some cases may continue to recur for several days after the febrile access. In the "adynamic" form complications with heart and lung diseases are not uncommon. The few cases of gangrene of the lungs after pneumonia, which I have seen, occurred in such drunkards. The course is always tedious, and the process of absorption especially protracted. Sequelæ are frequent.

The complication of pneumonia with *malaria* is one of a group of complications in regard to which it is exceedingly difficult to form a positive opinion.

Grisolle[1] treats the subject very thoroughly, and, as it seems to me, with much good sense and acuteness. The original articles quoted by him are not accessible to me, and I must therefore confine myself to only a brief statement taken from his work.

In the first place, there are two forms of this complication, which must be distinguished from each other—a simple coexistence of croupous pneumonia and intermittent fever, and a malarial infection localizing itself in the lungs. In the first case there is a mere complication, the symptoms run their course alongside of each other, and when the intermittent fever is cured by quinine, the pneumonia, which still remains, continues its progress undisturbed. The malarial infection, localizing itself in the lungs, exhibits itself, according to Grisolle, as either intermittent or as remittent pneumonia, and both belong to the group of pernicious forms.[2] These cases are characterized by the occurrence of pain in the side and bloody expectoration, coincident with the ushering in of the attack by an unusually severe and long-continued chill; at the same time the physical examination reveals dulness of the percussion note, and fine, dry, abundant crepitant râles, sometimes mixed with blowing sounds. These local symptoms continue through the hot stage of the intermittent, and then quickly subside, together with the fever, during

[1] L. c., pp. 411–423.

[2] *Griesinger*, Infectionskrankheiten, II. Auflage, p. 54.

the profuse sweating, at least in the first and second attacks. Occasionally, however, a slight amount of pulmonary lesion can be detected even during the apyrexia, which is said to be complete. The tertian and quotidian types are more frequent than the quartan. If the return of the attacks is not prevented, the patients commonly die in the third or fourth paroxysm; in many cases both lungs are affected, and severe brain symptoms are also present. The remittent form is distinguished from the intermittent by the fact that, during the remission a resolution of the local process does not always take place. Otherwise the distinction is not easy; the confusion between the two varieties is much greater than in the first-mentioned form. In both forms the whole disease, including the pulmonary affection, may be controlled, and perfect health restored, by the use of quinine.

It appears to be fairly well proved that a severe disease of the lungs may be produced by malarial poison, but it is by no means demonstrated that this affection is croupous pneumonia. The rapid disappearance of the local symptoms has repeatedly been urged as an objection. The way in which Grisolle sets aside this objection is perhaps a little too *ex cathedra;* he attaches but little importance[1] to autopsies which are said to have revealed changes identical with those found in "legitimate" pneumonia, but he still strongly insists that the results of physical examination are perfectly satisfactory. Until this pathological question is settled by satisfactory proof, I beg leave to at least express a doubt in regard to the actual existence of a croupous pneumonia.

Still another complication of pneumonia is the inflammation of the *parotid gland.*

I have never seen this complication; in the reports of the Vienna hospitals it is only mentioned six times out of 5,738 cases, a little more than one per thousand. The Basle reports do not mention it at all, nor does Huss. Grisolle speaks of it as a fortunately rare event; the patients most apt to be attacked are said to be those beyond sixty years of age.

[1] The authority for this statement is not given; in another place an autopsy by Dr. Catteloup is referred to, which showed "red hepatization." The same Catteloup is, however, censured by Grisolle on another occasion, because he has represented pneumonia and malaria as complications which are independent of each other.

According to Grisolle the parotid gland on only one side is usually attacked, but then in its whole extent. The inflammation progresses rapidly, and terminates generally in suppuration, or even in gangrene, for there is usually a diffuse infiltration of pus. For this reason the complication is always a serious one.

In our remarks upon the complications of pneumonia, mention has been made of the differences in the course of the disease which are thus produced. But little remains to be added. For in dividing pneumonia into forms or species for the sake of a correct nomenclature, it is much the same as it is with roses; either we call each individuum a species, or we admit only a single species. There are, however, some points of practical importance, which still remain to be considered. Many physicians are unaware that *abortive forms* of croupous pneumonia sometimes occur. Wunderlich[1] gives the following description of the course of the fever in such cases, and his account agrees perfectly with my own experience: "In one form there occurs, generally with a rigor and in an abrupt manner, a more or less considerable elevation of temperature (even to above 105.8° F.), which is immediately succeeded by a rapid defervescence, so that the normal temperature is restored by the second or third day. In a second series of cases the rise of temperature is more gradual, and somewhat remittent. The highest point (scarcely 104° F.) is not attained before the third day of the illness. The temperature then begins to decline in much the same manner as it rose." I have seen the first form more frequently than the second, especially in practice among children. The local affection may be slight, but by no means necessarily so. In some cases I have seen infiltration of the whole lower lobe occur also in adults, where in less than seventy-two hours the unusually high fever had entirely disappeared, and resolution took place very rapidly afterwards. In such instances, the great extent of the local affection should induce us to regard the case rather as one of an unusually mild course than as belonging to the abortive form, which term is more appropriate to smaller infiltrations.

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[1] Eigenwärme, l. c., p. 353.

I have repeatedly noticed that a number of cases of short duration are apt to occur together, just as in a particular epidemic it is by no means uncommon for the crisis to fall upon the same day of the disease. When the majority of the cases run this rapid course, there will be ample room for error on the part of those who have faith in the efficacy of abortive modes of treatment.[1]

What are we to understand by typhoid, or, as it is also called, asthenic, adynamic, and nervous pneumonia? Liebermeister[2] gives the following as characteristic symptoms: "The mild form ordinarily begins suddenly with a strong rigor, the pneumonic sputa appear very early, and the infiltration can be detected by physical examination; in the malignant asthenic form, on the other hand, we generally observe a gradual lingering access, the characteristic sputa of pneumonia are often absent, or, in place of the usual bright red or rusty brown color, the expectoration is dirty brown. Even early in the disease, the symptoms on the part of the central nervous system assume so much prominence that this form is often, from the beginning, mistaken for typhus or meningitis; at the same time the infiltration of the lung generally progresses slowly. In regard to the pathological anatomy, the severe is distinguished from the mild form by the far greater frequency of pleuritis, jaundice, and albuminuria. Furthermore, the character of the infiltration is different in the two forms; in the mild variety the lung on section presents a dry, firmer, and distinctly granulated surface, while in the severe form the surface is less markedly granular, is covered with a greasy, dirty brown fluid, and the lung tissue itself is usually quite rotten. This latter form is especially characterized by the frequent occurrence of an enlargement, which is often considerable, of the spleen, and by a pulpy condition of the substance of the organ." The form of pneumonia here described seems to occur far more frequently in Basle than elsewhere; in fact it appears to be endemic there. Every physician who has had much experience has met with it in isolated

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[1] See also *Lebert*, l. c., p. 595.

[2] Fismer, l. c., p. 443.

cases; at least we find it treated at more or less length by writers.[1] The symptoms are described in essentially the same manner by all of them. Liebermeister, however, definitely puts the question, which has at various times been obscurely hinted at, whether there is not present in asthenic pneumonia an etiological factor different from that existing in the ordinary form of the disease. Without giving a very positive answer, he is inclined to suppose that there is. I am of a different opinion, and for the following reasons. Leaving out of the question Basle, where the disease is endemic, and the isolated epidemics[2] in which this form is especially frequent, we find writers, myself included, unanimously agreeing that "typhoid" pneumonia occurs in persons who are from some cause or other in a weakened condition. Hunger, abuse of alcohol, previous or still existing diseases, excessive bodily or mental exertion, advanced age, and unusually bad hygienic conditions, are the causes most commonly assigned. For the production of asthenic pneumonia it is also generally necessary that there should be a diminished power of resistance to disease. I regard croupous pneumonia as on all occasions an infectious disease, and will give my reasons for this view hereafter. Entertaining this view I prefer to suppose that the endemic form at Basle (a city which has been grievously visited during the middle ages and in later times by malignant epidemics, and where an unusually severe typhoid fever is just now prevailing) and the epidemic form in other places are both due to an unusual intensity of the infecting principle. The distinction between this and the ordinary form of pneumonia is, therefore, in my opinion, only quantitative and not qualitative. On this supposition we can explain also the more frequent occurrence of complications; at least we see satisfactory analogies in the affections, which are acknowledged to be infectious. Repeated conversations with my friend and colleague Liebermeister have, moreover, convinced me that he regards my view as one that will bear discussion. It should not

[1] *Stokes*, A Treatise on the Diagnosis and Treatment of Chest Diseases; *Grisolle*, l. c., p. 336 et seq.; *Lebert*, l. c., p. 600; *Huss*, l. c., pp. 54, 55; *Wunderlich*, Handbuch, III., 2, p. 349.

[2] See *Grisolle*, l. c.

be forgotten that this form may be directly produced also by accidental complications.

The so-called cerebral, or latent form of pneumonia, will be considered in the chapter on diagnosis.

What are we to understand by relapses of pneumonia? The term relapse cannot be defined *à priori;* we can, therefore, only take the meaning which has been generally agreed upon. No such agreement has hitherto been made—probably it never can be without a "*sic volo, sic jubeo.*" If we take the anatomical lesions as the standard for comparison, as Griesinger has done for typhoid fever, and call the different stages of development of the pathological changes relapses, then we shall logically be compelled to regard as relapses every instance where different lobes are affected, one with red and another with gray hepatization. Almost all multilobar pneumonias would also come under this head. But with this use of the term it is not quite clear why we should not also regard as relapses the different stages of development of the inflammation in one and the same lung, especially if the differences between them are well marked. Nor does clinical observation afford us a logically justified point of distinction. The best plan, I think, is to let every one use the term relapse as he chooses, and to hold fast to the fact, that when a person has had one attack of pneumonia he is in danger of sooner or later having another.

As an illustration of the indistinctness with which the expression "relapse" is used, Fismer mentions, among his fatal cases, one (males, XII.) in which the first pneumonia of the right lower lobe had run its course, and thirty-nine days afterwards, when completely convalescent, the patient was again attacked and died from pneumonia of the whole left lung. The second case (males, XX.) was a patient who was taken ill on January 23d, with pneumonia of the right upper lobe; on the 29th inst., "when his general condition had already begun to improve," the disease extended to the two other lobes of the same side. In both instances he speaks of a "relapse."

Abscess of the lungs is regarded by many writers as a rare termination of croupous pneumonia. All physicians of experience regard exhaustion, from whatever cause, as specially predisposing to this complication. Great importance is attached also to a wide extent of the local affection.

All writers agree that abscesses are formed in pneumonia more readily in the upper than in the lower lobes. The course of a pneumonia which has this termination is not different from usual at the outset; but the favorable crisis does not occur at the expected time; the fever continues, the local symptoms do not resolve, and severe cerebral symptoms and attacks of cardiac insufficiency make their appearance. In many cases a diagnosis is impossible. It can be made only when the abscess has burst and left a large cavity filled with air, communicating with a bronchus and situated near the surface. Under these circumstances the signs of a cavity may be found. But we should be careful not to diagnosticate an abscess of the lungs by the physical signs of a cavity alone. I have previously mentioned how frequently a considerable condensation of the upper lobe will give rise to Williams's tracheal tone; the physical conditions being of such a character that on auscultation we might be led to infer the existence of a cavity. To make the diagnosis of a pulmonary abscess certain, we require, therefore, additional evidence, and this we obtain by the examination of the sputa, which contain large quantities of true pus mixed with characteristic portions of lung tissue (elastic fibres), and frequently with blood. When the expectoration presents these abnormal characters we may safely infer the existence of a pulmonary abscess, even when for a long time we are unable to detect its situation. In addition to these positive signs there is another one, which occurs very frequently, and which points with at least a certain degree of probability to the occurrence of this complication. I refer to the change of color of the sputum. Traube[1] was probably the first to call attention to this fact. In suppurating croupous pneumonia the expectoration is colored green by a pigment, which presents no reaction in the presence of nitrous acid, and can thus be easily distinguished from the coloring matter of the bile, which passes into the expectoration in complicating icterus. The color referred to is, however, by no means pathognomonic, for, according to Traube, it is found also "in cases in which no crisis takes place, but the fever subsides by

[1] Gesammelte Abhandlungen, II., p. 699 et seq.

lysis, and resolution is still possible."[1] It is also found in the beginning of caseous pneumonia running a subacute course (Traube).

The coexistence of pericardial effusion with the pneumonia was probably only accidental in Traube's case, which is described at the place referred to; and the same is true of another of Traube's published cases,[2] as well as of one observed by myself. I can corroborate the statement that the green coloring matter is not identical with an oxidation stage of the bilirubin.

Abscesses of the lungs may heal by adhesion of the walls and obliteration of the cavity. The pus may be discharged in any direction, wherever the resistance is least. Perforation into the bronchi is the most common; next in frequency is the escape into the pleura. The course of a pulmonary abscess is probably always a protracted one. Many cases have been recorded where healing was incomplete, resulting in the formation of a suppurating cavity with habitual expectoration. In regard to the ultimate result, writers differ.

Laënnec[3] asserts that out of twenty cases he has had only two deaths; the frequency of the occurrence of abscesses (all of them in the year 1823) he refers to a special "*constitutio morbi.*" With only two exceptions the abscesses are said to have healed in from fourteen to forty days. With regard to these observations, which are based almost exclusively upon the results of physical examination, a certain amount of doubt is permissible. Few persons will be so willing to swear in *verba magistri*, as Grisolle[4] has done on this occasion. The number of reliable observations is so small that it is impossible to form a positive opinion based upon statistics. Huss states that he lost twelve out of twenty cases of abscess of the lungs.

Gangrene of the lungs is also an uncommon result of croupous pneumonia. It occurs most frequently in the pneumonia of drunkards. Whether prostrating causes in general can be properly regarded as predisposing to this complication is undecided. The most probable anatomical cause is the occlusion of a vascular district in the lungs. But this factor alone

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[1] L. c., p. 700.

[2] Gesammelte Abhandlungen, II., p. 467.

[3] L. c., I., p. 321.

[4] L. c., p. 331.

cannot produce gangrene, as has been shown by the artificial production of embolism, as well as by clinical observation. Something else is necessary, a something which we might as well permit to sail under the neutral flag "infectious." The conditions which favor decomposition are nowhere present to so marked an extent as in the lungs. And yet the fact that the gangrene is, as a rule, limited to a small portion of the lung, shows that some other factor is involved, and that we have no right, without further evidence, to regard the whole process as a simple decomposition analogous to what takes place outside the living body. *Circumscribed* gangrene of the lungs, resulting in the formation of a cavity and limitation of the destructive process by reactive inflammation, occurs after pneumonia more frequently than the *diffuse* form, which extends over larger portions of the lung. In the anatomical diagnosis of pulmonary gangrene complicating pneumonia, we should be careful to examine whether the case is really one of croupous pneumonia. Laënnec[1] has insisted upon this point, but in a somewhat different form; he says that the inflammation produced around the gangrenous part appears to be the result rather than the cause of the mortification. Such a possibility is admitted in the case of putrid bronchitis, which has extended to the lung tissue. The symptoms of the pneumonia are not changed in any characteristic manner by the occurrence of gangrene of the lungs; the fever continues, and an adynamic condition is developed. These ambiguous symptoms become significant as soon as the sputa are found to contain characteristic fragments of lung tissue, which have undergone decomposition. Traube[2] strongly insists that elastic tissue is not present in the discharged gangrenous shreds of the parenchyma. There is no unanimity among writers on this point, and I must suspend my own opinion. Our knowledge of the other morphological elements, and of the chemical decompositions which have taken place in the discharged sputa is derived from the investigations of Leyden[3] and

[1] L. c., p. 349.

[2] Gesammelte Abhandlungen, II., p. 454.

[3] Volkmann's Sammlung klin. Vorträge, Bd. I., p. 195 et seq., and Deutsches Archiv f. klin. Medicin, Bd. II., p. 488 et seq.

Jaffé. In most cases a penetrating offensive odor betrays the existence of gangrene of the lungs even before there is any expectoration. To this rule, however, the exceptions are not very rare; my own experience in numerous cases agrees with the statements of other writers that sometimes large portions of the lung may be gangrenous without the presence of any offensive odor. For the recognition of this odor it is necessary that there should be a communication with the bronchi, and such a communication does not always exist. Under some circumstances a cavity may be detected as a result of the gangrenous process. The gangrenous fluid may escape by rupture into the surrounding parts.

Pulmonary gangrene may terminate in recovery; but this result is not to be expected if the gangrene be extensive. Huss lost ten cases out of twelve, and the records at Basle show that all of the eight cases which occurred there died.

Phthisis and *cirrhosis* are not very frequent results of croupous pneumonia.

In regard to the question recently propounded by Buhl, whether in case of such a result there has not always been a mistake in diagnosis, I must refer for the main part of my reply to what has been said in the section on pathology. But besides this, it is necessary to come to an understanding here in regard to the limitations of the expression "chronic pneumonia," which is frequently used by writers as a common name for both of the above-mentioned diseases. This precaution is indispensable if we wish to avoid disputing about trifles. Grisolle,[1] for example, includes as forms of the disease which are transitional to chronic pneumonia only those cases in which there has been a history of severe constitutional disturbance in connection with rapidly progressing changes and destruction of the lung. According to his view, a lung which has for months been less permeable to air than is normal, should not be regarded as affected with a chronic inflammation, so long as there is no constitutional disturbance. This statement is, in my opinion, somewhat too dogmatic; after a pleuritic exudation, which

[1] L. c., p. 338 et seq.

has permanently prevented the expansion of the lung, it is not uncommon, even without any objective disturbance in the lung, for an interstitial proliferation of the connective tissue to take place, which we must certainly regard as inflammatory. In these cases it is in fact the rule, rather than the exception, for the patient to feel perfectly well, and yet, in a pathological sense, an inflammation does exist. On the other hand, it seems to me that neither from a logical nor a practical standpoint are we justified in making an essential distinction in favor of those cases of interstitial pneumonia which arise after a croupous pneumonia accompanied by a pleuritic exudation. The *primum movens* is and remains the same factor which produces the croupous pneumonia; from the croupous pneumonia comes the pleuritic effusion, and from the pleuritic effusion the interstitial pneumonia, and finally the phthisis. The middle terms are different, the course of the pathological process is different; but we must still regard even this form as the result of the croupous pneumonia.

The picture of this form of the disease can be drawn only in the most indefinite outlines, because our observation of such cases is restricted to isolated instances. When a croupous pneumonia fails to resolve at the proper time, a period ensues which presents either local or general symptoms of a disturbance, and generally both together; in fact, all the stages, from the slightest variation in the process of resolution to the symptoms of an acute phthisis, which proves fatal within a few weeks. It is a mere arbitrary assertion to ascribe the origin of "chronic pneumonia" to any particular stage.

The mortality of croupous pneumonia is ascertained with probably more difficulty than that of any other disease, because there is no agreement whether we are to include the so-called secondary pneumonias which accompany other affections as their final result. Nor is it easy to advance any convincing argument whether this should be done or not. If we say that the anatomical condition should decide the question, then we must take into account all the facts of the case. It may be fairly objected that we ought not to place in the same category a croupous pneumonia occurring in a cancerous patient, and a croupous

pneumonia attacking a robust person. But if we admit such a principle of distinction, we throw the door wide open for the arbitrary expression of individual opinion, and are less likely than ever to get at the truth. Errors will creep in, however exact we may try to be, when the question turns upon the statistical proof of some mode of treatment. The best plan, therefore, is to admit all cases which were treated as croupous pneumonia, or in which death is said to have resulted from this cause, but at the same time to give the data for the clearly cut, uncomplicated cases. At least, if any complication occurred, it should be mentioned in the mortality report. This is not usually done, at least not in a logical manner; hence the uncertainty of mortality statistics.

Among the articles which have been written upon this subject, I wish to refer to one by Dr. Brandes, of Copenhagen,[1] more particularly, because a statement extracted from it by some one has been flitting about like the Wandering Jew, in a very dilapidated condition. Brandes does state, it is true, that with the same treatment he lost in one year 5.4 per cent., and in another 31 per cent., of his cases; but he also gives the reason, viz., that in the fatal year the cases presented numerous complications. This fact is not mentioned in the extract referred to, which has long been in circulation, and which to every intelligent person must at the very first sight have appeared ridiculous. Brandes states the fact thus:

1856...55 cases of pneumonia—3 deaths—complications not mentioned.
1857...87 " " 27 deaths.

Among the deaths: 12 cases of delirium (D. tremens).
5 complicated with typhoid fever.
5 " " organic heart disease.

Comparison of uncomplicated cases—
1856.....55 patients—3 deaths.
1857.....65 " 5 "

The difference in the mortality is so slight that, with these small figures, it is hardly worth speaking of; at any rate there is no occasion for making so much ado about it.

In pneumonia, hospital statistics are of as little value in regard to the percentage of mortality as in regard to the percentage of morbility. I have already shown at some length the

[1] Virchow's Arch., Bd. XV., p. 210 et seq.

reason for this in my remarks on the etiology, and shall at present only call attention to the fact that the two factors which strongly influence the mortality, viz., age and sex, are represented in very different proportions in hospitals and in the general population. In all statistics, therefore, the errors are so serious, and it is so impossible with our present data to estimate their magnitude, that but little reliance can be placed upon such figures.

I give a few compilations for the sake of complying with the established custom.

Vienna, General Hospital, 1858–1870.
7,942 cases of pneumonia with 1,944 deaths, = 24.5 per cent.

Stockholm, Seraphim-Lazareth, 1840–1855.
2,616 cases of pneumonia with 281 deaths, = 10.7 per cent.

Greifswald Polyclinic in ten years.
941 cases of pneumonia with 102 deaths, = 12.1 per cent.

Basle Hospital, 1839–1871.
922 cases of pneumonia with 213 deaths, = 23.1 per cent.

In Vienna, it appears, all the cases were included in which croupous pneumonia was found at the autopsy; for in the annual reports cholera, dysentery, peritonitis, etc., are mentioned among the complications. In Stockholm, a singular method of computation has been used. Only those cases were included in which pneumonia was present at the time of reception; those in which the disease developed during their residence in the hospital are excluded. Huss[1] makes the express statement that "if a patient with pulmonary tubercles were admitted with inflammation of the lungs, the case was recorded under the head of pneumonia; but this was not done if the inflammation of the lungs were developed after a longer or shorter stay in the hospital. The same rule was observed in regard to Bright's disease, organic disease of the heart, and other chronic affections, which predispose more or less to pneumonia." Many others will ask, like myself, why such an unnatural distinction should have been made. In the Stockholm reports there is still another fact which forbids our comparing them with other statistics. Some of the patients, who died twenty-four hours after admission, were not included. Huss uses this language: "Patients, who were admitted into the hospital after the hour of reception, and who died before the hour of reception (registration) of the following day were not registered among the patients, but were recorded among the 'unregistered deaths.'" Suppose, for instance, that the "hour for registration" lasted from eight to nine o'clock A.M.; then a patient who is admitted at eight o'clock, and dies at ten o'clock, is counted, but not one who is received at ten o'clock and dies at twelve

[1] L. c., p. 60.

o'clock. Why? If these non-official deaths be included, the percentage of mortality rises to 13.8. The Stockholm reports include only the ages from eleven to seventy years. The Basle reports are very instructive in more than one respect, especially in regard to the necessity of caution in estimating the percentage of mortality when the number of cases is small.

A division is made into three periods of 10, 9, and 9 years each, the total number of patients being respectively 223, 197, and 232. In single years the mortality varies from 5.9 to 33.3 per cent.; in these two instances the number of patients was seventeen and fifteen per year. That these marked differences were merely accidental is evident from the fact that in the three periods mentioned above, where larger numbers are involved, the average mortalities were 24.7, 24.9, 25.9 per cent., the percentage being about the same in each period. Fismer's reports are so exact that it is possible to discover the causes of the high mortality, at least for a total of 230 cases. One of the chief causes is the advanced age of the patients; nearly 19 per cent. of all the cases of pneumonia at the Basle hospital were over fifty years of age, while in Stockholm only 6 per cent. had attained this age. The analysis of 38 fatal cases,[1] according to the record of autopsies, shows that among the male deaths without complications, the youngest patient was fifty-six years old, and three were over seventy; all the other deaths of males were from complicated pneumonia, and in the great majority of cases from complications with diseases of long standing. The deaths among women show very nearly the same facts. The mortality statistics of the Basle hospital can be satisfactorily explained by the special nature of the material; Fismer's hypothesis, that the form of the disease was especially malignant, is unnecessary. Whether pneumonia be not generally a severer disease in Basle than it is elsewhere, is, of course, another question; at all events, Liebermeister's success in treatment has shown that these adynamic forms can be successfully managed, and hence his relatively favorable proportion of mortality, 16.5 per cent.

In general, we may say that when croupous pneumonia occurs in a hitherto vigorous individual, who has not been weakened by age, excess in eating and drinking, or other diseases, it is the least fatal of acute affections. But when it attacks persons with feeble powers of resistance to disease, the mortality is very great. These points will be considered more in detail under the head of prognosis.

Diagnosis.

In order to avoid repetitions, I shall confine myself to essential points.

In the great majority of cases of pneumonia, when the attack

[1] Compare details subsequently given.

begins acutely with a rigor, or at least with chilliness, a rapidly developed high fever, pain in the side, and dry cough with sputa of a characteristic color, these symptoms make a physical examination of the lungs imperatively necessary in order to ascertain the seat of the disease. When this is discovered the diagnosis is certain. Moreover, the form of the inflammation can be established anatomically by the discovery of fibrinous casts of the smaller bronchi in the expectoration, and clinically by the course of the temperature curve, and its rapid sinking to the normal, or below the normal, while the local process at first seems still to continue unchanged. If the results obtained by these methods of investigation are positive and constant, the diagnosis is as certain during life as at the autopsy; but the examination is not always of this fortunate character.

In the first place, it may be laid down as an incontrovertible proposition that there is no sign pathognomonic of croupous pneumonia. Neither the rusty-brown expectoration, even when fibrinous casts are found in it, nor the inspiratory crepitant râles are signs of such a character. An absolute reliance upon any one sign argues imperfect knowledge on the part of the examiner. Grisolle still continues to make this mistake.[1] The narrowness of such a view is shown by the analysis of the symptoms, which I have previously given. On the other hand, it is an excessive rigorism to reject the existence of pneumonia because all the signs are not present. Every one must draw the lines for himself, and whether he will succeed or not will depend upon his natural ability and the amount of his experience.

Two contingencies remain to be considered. Sometimes days will elapse before a central pneumonia has come sufficiently near the surface of the lung to be recognized by a physical examination. Even a skilful physician may, for a time, mistake such a case for one of the typhoid diseases, especially if the outbreak of the attack be preceded by an unusually protracted prodromal stage. It is difficult to lay down general rules for such cases. If the patient have had a chill, pain in the side, rusty sputa, and high fever, there is scarcely any room for doubt, and even in central pneumonia these symptoms are generally present.

[1] L. c., p. 471.

But when the only symptom is the sudden occurrence of high fever, the diagnosis can be made only from the consideration of all the facts presented by the history and present condition. The change in the pulse-respiration ratio is, in my experience, the most constant of all the signs, and therefore, in the absence of any other affection of the heart or lungs, the most certain. Much will depend upon the acuteness of one's senses. A skilful physician, with good hearing, will detect the seat of a pneumonia at a time when an inexperienced person is unable to detect any local changes. A slight abnormity in the percussion note, an altered resistance at this or that spot, a single deviation from the normal auscultatory sounds may, in doubtful cases, decide the question in favor of pneumonia. Still the place for the discussion of these modalities is the bedside, not a handbook.

The cases of pneumonia of the apex, with severe cerebral symptoms, which are seen most frequently in childhood, present less difficulty in diagnosis than is commonly supposed. They are most apt to be mistaken for meningitis. But a meningitis which is neither tuberculous nor traumatic, and is not produced by caries of the petrous portion of the temporal bone can, in my opinion, only be epidemic cerebro-spinal meningitis. It is therefore only with regard to the latter affection that a difficulty in the differential diagnosis can arise, because the two other forms are easily recognized by their history. With reference to epidemic meningitis, it is, of course, a question of the first importance whether any similar cases of disease have occurred previously. Furthermore, one or more points of the spine will be found to be so sensitive that when pressure is made the face of the patient will usually be distorted, even during profound coma. The pulse-respiration ratio should also be carefully examined. In a doubtful case we may be guided by the results of treatment. In cerebral pneumonia the brain symptoms depend essentially upon the fever; antipyretic treatment, the simple withdrawal of heat by cold water being the best plan, will reduce the temperature, and with this reduction the alarming symptoms will disappear. Cold affusions of the head and back are important subsidiary measures. Within twenty-four hours at farthest, after the employment of this energetic treatment, all the symptoms yield.

This is, of course, not the case in cerebro-spinal meningitis; the functional derangement of the brain here depends upon organic lesion, and is modified but not removed by the withdrawal of the fever, which is only one of the morbific causes. Moreover, it will often be possible, even at this early period, to discover enough local signs to confirm the diagnosis. Far more difficult than the distinction from meningitis is the differential diagnosis between pneumonia of the apex, with cerebral symptoms, and the hyperpyretic initial stage of the acute exanthemata, especially if the eruption be delayed, and there are no decided local prodromal symptoms. In such instances the general circumstances of the case, such as the prevalence of an epidemic, the diminished susceptibility to the contagion in question in consequence of a previous attack, etc., are more conclusive than all the symptoms taken together. Those forms of pneumonia which produce but little constitutional disturbance, as in aged, cachectic, and very fat persons, require a thorough examination of the thoracic organs. Perhaps the first thing which attracts attention to the lungs is the dyspnœa. In every case of delirium tremens, with fever, it is advisable to make an examination of the lungs if one wishes to escape unpleasant surprises at the autopsy. The same remark applies to children who are teething, or are supposed by the parents to be "suffering from worms;" the physician who neglects the warning of the thermometer in such cases will often have occasion for regret.

It is hardly necessary to discuss here the details of the differential diagnosis between croupous pneumonia and pleuritis. I shall content myself with again calling attention to the very great significance of the signs of displacement of organs, and shall refer the reader to what has previously been said on this subject. The differential diagnosis from catarrhal pneumonia will be considered in the next article.

Another affection requires to be mentioned, which Buhl has recently wished to introduce into pathology under the name of genuine desquamative pneumonia. Its situation in the upper lobes, its progress from above downwards, and especially the character of the sputa, which Buhl regards as characteristic; the very large amount of alveolar epithelium, ciliated cells, myeline

degeneration of the alveolar epithelium, and free myeline in the sputa; these indications are said to be amply sufficient for the diagnosis. Urgent dyspnœa, cyanosis, cough, and high fever accompany this form; death is the usual termination, and results from fatty degeneration of all the organs, especially the heart.[1]

My own experience with the affection in this locality has been so small that I am unable to give an opinion in regard to the statements which Buhl has made.

I have said so much upon the subject of diagnosis, in my remarks upon individual symptoms, that it is unnecessary to continue the subject at farther length in this place.

Prognosis.

I begin by grouping the facts, which experience has shown to bear upon the mortality of croupous pneumonia, and then from these individual factors I shall attempt to ascertain the common quantity, the fundamental condition upon which the mortality really depends.[2]

1. *Time of Life.*

a. From Childhood to Puberty.

During this period, if the child have been hitherto vigorous, the danger from pneumonia is less than at other ages. Feeble children succumb to this disease during the early years as easily as individuals of more advanced age.

The older writers, who were not aware of the distinction between catarrhal and croupous pneumonia, regarded childhood as a very fatal period; and it certainly is, if everything that is called pneumonia be included. But when the necessary distinction is made between the two forms of disease, this opinion can no longer be maintained. Out of 201 cases of croupous pneumonia up to sixteen years of age inclusive, the great majority being under ten years, Ziemssen lost only 7 (3.3 per cent.). In my experience during two years at the Kiel polyclinic, with 110 patients under ten years of age, I lost only 4, and all of these died from complications. Barthez,[3] out of 212 children from two to fifteen years of age, lost only 2.

[1] Zwölf Briefe, l. c., p. 48 et seq.

[2] See below, p. 152 et seq.

[3] See Grisolle, l. c., p. 516.

Funck's statement in his dissertation that the deaths from "pneumonia" between one and ten years for the Greifswald polyclinic amounted to 10.3 per cent., shows that he probably confounded the croupous and catarrhal forms. This is evident, it seems to me, from a comparison of these statistics with those of Ziemssen, who in part used the same material. It is hardly worth while to make this comparison in detail. Pneumonia is very prevalent in foundling institutions and in the hospitals of large cities which are provided for very young children. Grisolle frankly admits that in these institutions pneumonia is almost necessarily fatal, at least that this is the case in Paris.

b. The Age of Vigor.

Pneumonia during this period also is not a dangerous affection. It is uncommon for uncomplicated pneumonia, in a person who has been previously healthy to prove fatal. To prescribe definite limits to this period, to confine it to this or that year is, in my opinion, inadmissible. It varies so much in different places that the quotas for separate years in different countries cannot be compared with each other, except during a very short portion of the lifetime. The main causes which impair a man's physical powers, and shorten the active period of his life, are excessive physical exertion and the immoderate use of alcoholic drinks. Hence it is impossible to compare the age classifications of a manufacturing population with those of the laboring classes of a North German seaport town, or with those of a strictly rural district. The porters of Kiel, for example, are usually old by the time they are thirty, while our peasants in many parts of the country are still hearty fellows at fifty. I insist strongly upon this point, because I wish to protest against the idea that there is necessarily an increased fatality after a certain limit of age is passed.

I will give a few details:—

Fismer lost 4 out of 97 patients from sixteen to thirty years of age; for the decade from thirty-one to forty years the percentage was considerably higher, 48 patients with 9 deaths. I use these relatively small figures, taken from Section A, because the same ratio is repeated in Fismer's Class B. In Greifswald the fatal time of life is about ten years later than in Basle. Out of 130 patients between thirty and forty years of age, Funck lost only 4 (3 per cent.). In Stockholm the thirtieth year forms the dividing line; the mortality from twenty to thirty years is only 5.9 per cent., while in the following decade it rises to 11.9 per cent. The absolute figures are 1,041 patients with 61 deaths, and 816 patients with 97 deaths.

I think these data are sufficient to justify my reservation

c. The Age of Decline.

During this period, pneumonia is one of the most dangerous diseases. The greater the wear and tear of the body the more fatal the disease becomes.

The age classes included in this period will admit of comparison with each other, at least this is the case for the same population. All the published statistics show that the mortality increases from year to year.

	40–50 Years.	50–60 Years.	60–70 Years.
Griefswald.....	9.5 per cent.	20 per cent.	37.5 per cent.
Stockholm.....	19.8 "	21.6 "	24.1 "

The absolute figures here given are widely different for the two places, but in each case the series ascends. In Paris about one-half of the patients beyond sixty years die: 129 patients, 77 deaths, = 59 per cent.[1] The same is true of Basle. Writers are so unanimous upon the main point that I refrain from giving any more figures.

2. *Sex.*

Pneumonia is, *ceteris paribus*, a more dangerous affection in the female than in the male sex, in about the ratio of three to two.

The greater mortality among women is shown by all the reports which include large statistics.

Vienna.....	5,467 men	with 1,149 deaths	(21.0 per cent.).
	2,475 women	with 795 "	(31.1 per cent.).
Stockholm..	2,259 men	with 291 "	(12.9 per cent.).
	451 women	with 84 "	(18.6 per cent.).

Further details upon this point are unnecessary.

The danger is increased in pregnancy, especially if miscarriage occur.[2]

3. As regards the prognosis in complications, heart disease in all its forms seriously increases the risk to life. This complication does not directly threaten any vital organ, as is the case, for

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[1] See *Grisolle*, l. c., p. 517.

[2] See above.

example, with the brain when the complication with meningitis occurs, and consequently the danger arises from the derangement of the heart's action, resulting from the intercurrence of the new disease. For details I refer to what has been said in the section on complications.

4. The morbific cause varies in the intensity of its action not only in different places, but also at different times. The prognosis will depend also, *ceteris paribus*, upon what, in infectious diseases, we are accustomed to call the severity of the epidemic.

This fact is shown by the statistics of both Vienna and Stockholm, and has such an important bearing upon our theoretical conception of pneumonia that I may be permitted to give the figures *in extenso*.

Stockholm.				Vienna.			
Year.	Cases of Pneumonia.	Deaths.	Percentage of Deaths.	Year.	Cases of Pneumonia.	Deaths.	Percentage of Deaths.
1840	112	19	17.0	1858	854	203	23.8
1841	103	15	14.6	1859	439	98	22.3
1842	138	15	10.9	1860	450	116	25.8
1843	137	22	16.1	1861	638	146	22.9
1844	98	10	10.2	1862	678	181	26.7
1845	141	26	18.4	1863	756	162	21.4
1846	142	22	15.5	1864	690	183	26.5
1847	198	20	10.1	1865	501	118	23.6
1848	183	23	12.5	1866	437	116	24.2
1849	257	43	16.7	1867	492	123	25.0
1850	161	18	11.2	1868	532	128	24.0
1851	251	24	9.8	1869	729	181	24.8
1852	211	34	16.1	1870	746	189	25.3
1853	153	24	15.7				
1854	163	22	13.5				
1855	262	38	14.5				
				In Stockholm there were 2,710 patients with 375 deaths.			
				In Vienna 7,942 with 1,944 deaths.			

In Stockholm the average mortality was 13.83 per cent.; the "extreme variation," that is, the maximum + the minimum variation, taking the average as 100, was 62.7 per cent.

In Vienna the average mortality was 24.5 per cent., the extreme variation only 21.6 per cent. This difference, irrespective of the greater number of cases in Vienna, seems to me to be clearly due to the fact that in that city, as has already been

mentioned, all the cases which were anatomically ascertained to be croupous pneumonia were counted, while in Stockholm, according to the plan adopted there, not a few cases of pneumonia occurring in the sickly were excluded.

The mortality of pneumonia is influenced not only by the intensity of the morbific cause, but also by "the resisting power of the patient." The sum total of deaths is made up partly of deaths due to a lack of resisting power, and partly of those cases in which, although the resisting power was normal, the morbific influence was strong enough to overcome the resistance. If the number of the first class be large, a fluctuation in the second would of course be less noticeable, and this is doubtless the case with the Vienna record as compared with that of Stockholm.

The number of patients treated in Stockholm and Vienna is sufficiently large to exclude accidental causes, and hence the difference in the mortalities must be referred to some underlying changeable condition, which, in my opinion, is to be found only in the varying intensity of the morbific cause.

The operation of such a cause may, it seems to me, be inferred also from certain other facts. It not unfrequently happens that a group of cases of pneumonia occurring at the same time will exhibit a remarkable uniformity in duration. I have repeatedly observed that during a given period all the cases terminate on the fifth or sixth day, or at another time after the ninth day. These facts are, to my mind, best explained by the hypothesis advanced above.

5. The more extensive the localization of a pneumonia, and the more protracted the constitutional affection, the greater, *ceteris paribus*, is the danger to life.

This proposition is so evident that statistical proof is unnecessary.

To pass now to special prognostic indications, the following may be regarded as favorable: moderate fever, not above 104° F. in the morning hours, spontaneous remissions, marked effects from antipyretic treatment, and a not too rapid defervescence.

A regular, full pulse, which does not average above 120.

Respiration not very frequent; the ratio to the pulse not greater than one to three. Deep respiration possible without too much pain. No continued paroxysms of cough. No extensive bronchial catarrh. Locally, the infiltration completed

within about thirty-six hours; no tendency to spreading; situation in the lower lobe. Expectoration entirely absent, or in moderate amount, rusty brown, and not changing in color.

On the part of the digestive organs no symptoms beyond those of simple febrile dyspepsia. Eruption of herpes. Cerebral symptoms only in a degree corresponding to the fever. Perfect health previous to the illness, and unimpaired power to resist disease. In women absence of pregnancy.

The unfavorable indications are the reverse of those just described.

Treatment.

It is customary to class croupous pneumonia among the local, and not among the constitutional diseases. The question is liable to be prejudged when stated in this indefinite way. I prefer to formulate it thus:

Do facts justify us in regarding the anatomical changes always found in the lungs in croupous pneumonia as the essential cause of the other symptoms, especially the fever and constitutional disturbance? Or, are the pulmonary lesions and the fever both due to a common fundamental cause? Is there also a specific morbific agent, which excites what we call croupous pneumonia as a result of its variable action upon an organism already predisposed to the disease?

That croupous pneumonia is not a contagious disease is universally admitted. It lacks one necessary characteristic; the strictly typical course, which is peculiar to most malarial affections, is not present in pneumonia so constantly and with such distinctness as in that class of diseases. Furthermore, pneumonia is sometimes aborted by energetic treatment. Thus a tradition has been formed, sanctioned by the authority of centuries. When dissection began to be practised, the solid inflammation of an organ so vital as the lung stood out in bold relief, and for a long time engrossed attention. The old cardinal symptoms of inflammation—pain, swelling, redness, and heat—were present, or at least there were some indications of them. Almost every writer has described the expired air in pneumonia

as very hot, but J. P. Frank[1] has been obliged to reject the value of this symptom.

It is thus easy to see how, under the influence of tradition, the principal stress was laid upon the inflammation of the lung, and the question of its constitutional nature quietly avoided. Avoided, I say, because only a superficial study of the older writers can induce us to suppose that when they spoke of "Febris pneumonica," they meant anything else than pneumonia with less strongly marked local symptoms. In the very clearly stated and strictly humero-pathological exposition which Huxham[2] has given in regard to the etiology of pneumonia, there is a similar avoidance of a definite presentation of the question.

My reply to the question is this:

Croupous pneumonia is a constitutional disease, and is not dependent upon a local cause. The pulmonary inflammation is merely the chief symptom, and the morbid phenomena are not due to the local affection. The hypothesis of a morbific cause is indispensable. Croupous pneumonia belongs to the group of infectious diseases.

The proof of these propositions involves the consideration of all the facts bearing upon the etiology, anatomy, and pathology of the disease.

The morbific agent cannot be isolated; we are obliged therefore to study the etiology of the disease first of all by comparison with other affections, which are known to be non-infectious. And for this purpose no one will object to the selection of the non-infectious form of bronchitis and pleurisy. These diseases have always been regarded as bearing a very close relationship to pneumonia, bronchitis being classed among the "inflammatory diseases," and pleurisy among the "diseases contracted from cold." It has been, and still is, the popular idea that it depends upon the condition of the individual whether he contracts a pneumonia or a catarrh. If two persons be subjected to the same exposure one will get a pleurisy, the other a pneumonia,

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[1] L. c., p. 122.

[2] Dissertatio de pleuritide et peripneumonia. Opera physico-medica, Tom. II.

according to the personal *locus minoris resistentiæ*. We now know that this supposed relationship does not exist.

Because,

1. Croupous pneumonia and bronchitis exhibit an entirely different geographical distribution. The laws which govern the geographical prevalence of bronchitis are not applicable to croupous pneumonia. The *spatial* behavior of the two diseases is entirely different.

2. There is also a striking difference as to the seasons of the year in which these diseases prevail. There is, therefore, no *temporal* coincidence.

3. Generally speaking, "inflammatory diseases" and "diseases of the respiratory organs" are not shown by statistics to coincide in point of time with pneumonia.

4. The cases of pneumonia in which there has been a previous exposure to cold, or other influences of an injurious character, are so few that it is hardly possible to regard these influences as exciting causes; and, on the other hand, only a very small proportion of the persons who have been thus exposed are subsequently attacked with pneumonia. With what shadow of right, then, can we infer a causal connection? It would be just as correct to ascribe a typhus fever to the dietetic errors committed by the patient who is suffering from the febrile dyspepsia of the prodromal stage of the disease, as to regard the previous exposure to cold as the direct cause of the pneumonia. The outbreak, or more properly the development of the pneumonia, may be hastened by such exposure, but there is no other connection. Fright may bring on labor in a pregnant woman, but it ought not to be held responsible for her pregnancy.

These are the most important points in regard to etiology.[1] The first three mentioned I regard as important only so far as they tend to dispel any belief in a relationship between pneumonia on the one hand, and pleuritis and bronchitis on the other, based upon their spatial and temporal coincidence.

Our positive knowledge in regard to the etiology of pneumonia is so limited that we must look for decisive proof on this question in another direction.

[1] For the proof, see the chapter on etiology.

Now the fact that the *anatomical* changes[1] in croupous pneumonia are entirely distinct from those of every other pulmonary inflammation is a telling argument. Croupous pneumonia can no more be produced by the excitants of inflammation than can the characteristic intestinal lesions of typhoid fever. In pleurisy and bronchitis the exciting cause, whatever its character may be, as soon as it reaches the necessary intensity, always produces anatomical changes, which vary only in amount. *Croupous pneumonia cannot be produced by any of the usual causes of inflammation, however strong or weak their action. As in typhoid fever, there must be a special exciting cause.*

There is much to be said from the standpoint of pathology, but for the sake of avoiding unnecessary details I shall confine myself to the principal arguments.

1. *During the whole course of pneumonia there is no constant relation between the local and the febrile symptoms, nor dependence of the one upon the other.*

It is a plain deduction from the laws of causality that a causal connection between two things cannot be predicated, unless quantitative changes in the one are accompanied by quantitative changes in the other. In pneumonia no such dependence can be traced. The smallest pneumonic consolidations often run their course with the severest fever, and, on the other hand, we find extensive inflammations with a very moderate fever. Small consolidations with high fever and severe constitutional symptoms, and solid infiltrations with a comparatively slight fever and general disturbance, this is the rule and not the exception. This fact shows the fallacy of the pretext that it is the *individual* conditions in these cases which exert the decisive influence.

The extension of the local affection to parts of the lung hitherto unaffected is often accompanied by an increase of fever; but this fact in nowise invalidates the proposition advanced above. Coincidence does not imply causality. Who would be willing to regard an eruption of small-pox occurring the second time in the same person as the result of the cuta-

[1] See chapter on pathology.

neous inflammation, or to admit that the fever of a relapse in typhoid fever is due to the intestinal inflammation.

The resolution of the constitutional symptoms, and especially the suddenness of their disappearance, afford us an additional proof.

What local change has taken place in the inflamed lung at the moment when the temperature falls? This we are in no position to demonstrate, but the difference between pneumonia and pleurisy, for instance, in this respect is striking. The usual causes of inflammation never induce a process so completely independent of the constitutional symptoms as we see it in croupous pneumonia. Days and weeks elapse in this affection before the local pulmonary symptoms disappear, and yet during the whole time the temperature is low, and the patient feels quite well. Perhaps a still more satisfactory illustration of this point may be found in the contrast presented by the course of catarrhal pneumonia, which is a form of inflammation of the lungs produced by the usual causes of inflammation.

To recapitulate:

We find in pneumonia (prodromal stage) fever without local affection, local affection without fever (regressive stage). The local affection and the fever do not usually correspond in amount; hence we are justified in concluding that there is no causal relation between them, but merely one of coincidence.

2. *Croupous pneumonia is a disease which runs a typical course. No affection which arises from a local lesion presents a career so definitely limited in point of time as is the case with croupous pneumonia.*

Intermittent fever, the acute exanthemata, the different varieties of the typhoid group, in short, all the diseases which we regard as due to the action of a specific morbific cause, are characterized by a regularity connected with certain days. So long as the morbific cause continues its sway, the laws established by it also continue in force. Now, besides these laws, the body is subjected also to the laws of vitality, and the coexistence of these destructive and conservant forces can be clearly recognized. When the activity of the morbific cause subsides, it only remains for the conservant forces to restore the normal condition.

Moreover, every disease caused by infection may be divided into two periods. In the first, during which the morbific cause dominates, there is a conformity to law which is not seen in other affections; and it is this which justifies us in speaking of types of disease. But as soon as the morbific cause ceases to act, this regularity disappears also, because now, during this period of restoration, the control is assumed by those factors which constitute what we consider as the strength of the patient's constitution. The duration of the restorative process, and the mode by which it is effected, consequently vary in an indeterminate manner.

In all diseases of local origin, such as pleurisy, pericarditis, bronchitis, and catarrhal pneumonia, no type can be recognized. During the incubation, at the height, and during the decline of these local affections, the vital forces of the patient rule the situation, just as in the restoration period of the acute infectious diseases.

The manner in which croupous pneumonia runs its course is closely allied to that of the acute infectious diseases.

I refrain from giving the evidence in detail, because it seems to me that the facts already mentioned are sufficient to class pneumonia among infectious diseases; at least they are quite as satisfactory as those which are urged in favor of such a classification for cerebro-spinal meningitis, for example. The objection that epidemic meningitis prevails only for a limited time, while croupous pneumonia is a permanent affection, is far outweighed by the fact that in the latter the morbid product has a special anatomical character. The prevalence of intermittent fever among all classes, throughout the whole year, does not disprove its infectious nature.

This chain of proof might easily be lengthened by indirect evidence, or by facts less generally known than those which I have used; but would the chain be made thereby any stronger?

The diseases which I should especially like to have classed in the same group with croupous pneumonia are acute articular rheumatism and epidemic cerebro-spinal meningitis. These three diseases are undoubtedly non-contagious, either directly or indirectly, and should be referred to the category of malarial infections.

I am the more desirous that croupous pneumonia should occupy its proper position, because the moment we are convinced that we are dealing with an acute infectious disease—a constitutional affection with local symptoms, and not with a local disease with constitutional symptoms—from that moment the indications for treatment are radically changed.

When we realize that we have to combat not an "inflammation," but rather a constitutional disease, and one, moreover, of comparatively short duration, we readily fall into an expectant treatment which bides its time, interferes only when necessity requires, and does not see in the mere name of the disease an indication for attack. If we are chiefly occupied with the idea of a "phlogosis," we almost necessarily associate with it the "antiphlogistic treatment." This conception, of course, permits us to individualize, but only quantitatively; for the very essence of such a conception is that the treatment as such is opposed to the disease as such, and the patient himself is a secondary consideration. In the therapeutics of the present day we are still suffering far too much from the traditional ideas which stamped every remedy commonly employed for a particular morbid process as a "method" against a "disease." Such a mode of thought necessarily leads to confusion in regard to the true object of therapeutics and to routine treatment. I was obliged to smile when I heard from a medical writer, who with a certain positiveness prides himself upon being a critic, the reproach that I brought into the field against pneumonia a "formidable apparatus of remedies." Certainly if I can save the life of a patient with pneumonia I have nothing to fear, not even the second Dietl by whom I am menaced. A misapprehension of this kind springs directly from the traditional folly which I have characterized above. When the treatment of croupous pneumonia is conducted on "principles" such as I have described elsewhere,[1] it is merely following out these principles to do almost nothing for say eighty per cent. of the cases, and to employ the most active treatment for the other twenty per cent. It does not follow that when a person adopts the principle not

[1] Volkmann, Sammlung klinischer Vorträge, No. 45.

to get drunk, he ought therefore to join a temperance society. It is desirable that such a fundamental distinction should be a little more respected.

The conduct of the physician at the bedside of a patient ill with croupous pneumonia forms the conclusion of my task. I shall not enter very much into details, because these are given in Köhler's work,[1] which is an almost exhaustive exposition of the subject, and to which the reader is referred.

If we regard croupous pneumonia as an acute infectious disease, the indications for treatment are very much simplified. *Nature cures, and the only duty of the physician is to maintain life until this cure is effected.* This is the principle which underlies the management of all acute infectious diseases, and is applicable here unconditionally.

Can we succeed in cutting short or aborting the pneumonia, that is, in interrupting at any one moment the development of the disease? If we suppose that we are dealing with a local inflammation, we are justified in again and again searching for means by which to subdue the inflammation and abort the disease. For in other forms of inflammation—as, for instance, peritonitis—this may be actually accomplished. Hence the attempt to diminish the severity of the inflammation by early bleeding, a mode of treatment which has always in the past been regarded as indispensable. It was supposed that there was a local inflammation to be combated, and that for this purpose the abstraction of blood was the appropriate, nay, the sovereign remedy. Taking this view of the case, there certainly was a demand for energetic bleeding; and when Bouillaud took as much as ten pounds of blood before he saw the patient recover, we must at least acknowledge that he had the courage to follow his premises to their ultimate conclusions. If we admit the fact of the typical course of pneumonia, it seems to me logically impossible to recommend venesection for the purpose of aborting the whole process. To do so would be to join together two conceptions which have nothing at all in common. For this supposed abortive action of venesection we have no evidence

[1] Handbuch der speciellen Therapie, Bd. 1, p. 771 et seq.

which rests upon any other foundation than the crudest empiricism, especially in these days when our knowledge of the changes which take place in inflammation affords scarcely the slightest basis of support for an antiphlogistic effect from bleeding. The same thing may be said in regard to all other modes of treatment which are asserted to cut short the disease. They are and can be nothing else but empiricism. The empirical method asks, Does the introduction of the new factor change the normal course of things so uniformly that the alteration can be referred to the new condition? Tried by this test the worthlessness of the results of the abortive treatment is readily seen.

Out of every 100 cases of pneumonia, sixty-four normally terminate between the fifth and eighth days.[1] Kocher,[2] who ascribes an abortive effect to veratrine, found that in 70 per cent. of the cases defervescence occurred between the fifth and eighth days. In view of this fact, of what value is Kocher's statement that in 18.5 per cent. of his cases recovery took place between the second and the fourth day, while the average during this period is only 13 per cent. If the numbers involved were very large, this small difference would have to be regarded as significant; but Kocher's cases only number fifty-four in all, and the 18.5 per cent. referred to includes in reality only ten patients. A glance at the table (page 54) will show that equally large differences occur when not a particle of veratrine is given, proving that his results were undoubtedly accidental.

I have taken the most favorable of all the reports on this subject, in order to show how we may be misled by a lack of method. The objective study of facts is ever preaching to us the lesson of modesty. The effort to control disease is like trying to run our heads through a wall; the wall is generally harder than the head. The moment the "magister" is willing to unreservedly give up the place which he imagined he was able to occupy, to the "minister naturæ," our treatment will be more successful, in my opinion, than it is now. At all events, we shall then have heard the last of the "abortive" and all other "methods."

In studying the treatment of croupous pneumonia, the capital fact to be remembered is this: the illness, or, more correctly, the morbific force in croupous pneumonia is not a pernicious power

[1] See above, p. 55.

[2] Behandlung der croupösen Pneumonie mit Veratrumpräparaten. Würzburg, 1866.

which confronts the organic forces as a reckless destroyer. And this is true in both directions. Neither is the quantity of the morbific agent affecting the individual, as a rule, so overpoweringly great, nor is the power of resistance on the part of the patient usually so small as to produce an inevitably fatal result. Croupous pneumonia is distinguished from other, but not all acute infectious diseases, by the fact that it generally attacks persons who are already in an enfeebled condition. In other words, the danger to life from this affection arises far more from the defective power of resistance on the part of the patient than from the intensity of the morbific cause. This circumstance demands especial attention. In the plague and in diphtheria the danger depends chiefly upon the force of the infecting material. A mere reference to these instances makes the distinction clear. When the young and old, the strong and feeble, under similar influences, succumb to the same form of disease, we are justified in regarding the individual factor as the subordinate one. In these cases there is so little basis for the action of remedies, that the physician stands at the bedside rather as a hopeless spectator than as one who can restore health by his energetic interposition. Any one who has seen much of diphtheria knows full well his own weakness.

The first duty of the physician, in my opinion, is to inquire what does experience show to be the cause of death of pneumonia? Then comes the question, Is the case necessarily fatal, or is it possible to diminish the danger; in other words, is the derangement, which is called the "disease," of such a nature that life and death are bound together by it as with an endless and indestructible chain, or is there no such necessary connection? Are the morbid phenomena only such as can be overpowered as soon as we learn in what their destructive force consists, and where the point of attack of the disease lies? In the case of typhoid fever the inquiry into the point of attack has led us to oppose to the destructive force of this affection a dam, which in the majority of cases is able to withstand even the spring flood of the disease. Thus our treatment is being directed less and less to the causes of disease. The same thing is true of croupous pneumonia. If we analyze the morbid phenomena, which

are constantly present in this affection, we find that they consist of:

1. An interference with the function of the lungs.
2. Fever.

Neither of these conditions is fatal by itself. As soon as the falling temperature announces the crisis, the pulmonary affection becomes almost completely subordinate and insignificant, although at this time the functional disturbance in the lungs has to all appearance undergone but little change. The thorough analysis, previously given, of the course of the fever shows clearly that the increase of temperature in this disease is of such short duration and moderate amount that it is not sufficient by itself to cause death. These remarks, it is to be expressly understood, apply only to the great majority of cases. In some isolated cases, of course, the fatal result may be due solely to the extent of the infiltration, or to the height of the fever.

But if, with these exceptions, neither the fever nor the pulmonary affection is by itself sufficient to account for the danger in pneumonia, what then? Shall we search for a hypothetical third factor, or shall we try to solve the difficulty with the aid of the conditions at hand? I prefer the latter course.

Either of the two factors, it is admitted, is by itself insufficient to endanger the life of the patient; but the functional disturbance in the lungs and the fever are both harmful. It may easily be understood, therefore, that the united effect of both of these conditions might accomplish what either could not produce by itself. In this connection we have two possibilities to consider. Either the pulmonary affection and the fever have each a point of attack peculiar to itself, or they both have one in common.

I maintain that the latter alternative is correct, and can be proved to be correct. I advance, therefore, this proposition:

The danger in croupous pneumonia threatens principally the heart of the patient. Death results from insufficiency of the heart.

And now for the proof of this opinion.

1. *The exudation in pneumonia produces an increased resistance in the pulmonary circulation, and consequently increased effort on the part of the right ventricle.*

The exudation deposited in the lung exerts pressure upon the surrounding parts. In accordance with the law of impenetrability of matter it makes room for itself by driving out the previous occupants, the air in the bronchi and alveoli, and the blood in the vessels of the diseased section of lung.[1] This displacement can take place, however, only to a certain extent, because the pressure of the exudation is opposed by the forces which move the air and the blood in the lungs. The displacing force of the exudation, on the one hand, and the muscular force employed in carrying on the cardiac and respiratory functions on the other, enter the lists against each other. The amount of displacement depends upon the varying action of these three factors.[2] One thing, however, is certain; as soon as the equilibrium is established, the circulation of the blood through the diseased part meets with increased resistance. In the case of the pulmonary vessels lying outside of the inflamed section, there takes place what always takes place when, in a system of branching tubes, a portion of the escape tubes are closed, and it is necessary in a given time for the same quantity of fluid to pass through as in the open system. This can be accomplished only by increasing the impelling force. This illustration applies to the case in hand; more effort is required from the right side of the heart to drive the blood through the lungs, in order to prevent a retardation in the pulmonary circulation by the pneumonic infiltration. Moreover, the pressure of the exudation increases the difficulty of the left ventricle in nourishing the inflamed section through the bronchial arteries, though this additional burden is probably unimportant.

2. *The changes produced in and near the lung by pneumonia diminish the total amount of force to be furnished by this organ for the movement of the blood.*

The lung promotes the circulation of the blood by means of the respiratory muscles. In itself it is merely a passive organ. But in order that a portion of the vital force supplied by the respiratory muscles may be available for the maintenance of the circulation, it is necessary that the lung should be able to change its volume, to expand and contract again. If, however, a portion of the lung be infiltrated by pneumonia, it is no longer able to change its volume. And yet the portion of the power exerted by the respiratory muscles, which is withdrawn to this section of lung, remains the same, because these muscles act upon the thorax as a whole. But

[1] To complete the evidence, we may here mention the increase in volume of the diseased portion, which is found in every pneumonia. In the larger infiltrations it is not uncommon, as is well known, to find well-marked impressions of the ribs on the inflamed part.

[2] The conditions during the process of exudation are in fact very complicated, because a very considerable portion of the exudation comes from the blood-vessels, and is, therefore, subject to the same impellant forces as the blood. This subjection ceases as soon as the effusion coagulates, and it is to this period that the statements above made apply.

since the availability of the vital force supplied by these muscles for the circulation depends upon the expansibility of the lung, there must now be lost for the circulation just so much force as is transferred to the infiltrated section when its function is normal.

This waste of force caused by the altered physical conditions is still farther increased by a physiological factor which is rarely absent.

In the great majority of cases the pneumonia is accompanied by an inflammation in the adjacent pleura. Hence pain occurs as soon as the pleural surfaces are irritated by the respiratory friction. From the very outset the patient tries to make the friction as slight as possible by breathing superficially. But the necessity for breathing *sufficiently* is still more urgent. The diseased side cannot be kept quiet while the sound one is in motion, and so in most instances the patient instinctively accomplishes his object, as well as he can under the circumstances, by lying on the affected side, and thus increasing the amount of work it has to perform. He adds to the work of the muscles, which move this half of the chest, the task of raising the weight of a portion of his body. But the force necessary for this result is not applied to the half of the chest upon which the burden is thrown. This side is also less strongly expanded. The other methods, which are used by patients in pneumonia to alleviate the pain, such as firmly binding or holding the side, or bending the spine so as to approximate the ribs to each other, are all very similar in their action. This imperfect expansion of not only the diseased part, but also of the whole lung, on the burdened side, causes another loss of a portion of the vital force supplied by the respiratory muscles for the maintenance of the heart's action.

How completely this deficiency in the labor of carrying on the circulation by means of the respiratory muscles may be compensated by additional work done by them, cannot be determined *à priori*. But the greater the activity of the muscles, the greater their demands upon the force of the heart, because when a muscle works more vigorously it requires more blood; during its contraction the vessels are compressed, and there is an increase of resistance. This additional work affects the left side of the heart, and is consequently of less importance.

3. *In pneumonia the surface over which blood and air come in contact with each other is diminished by the exudation, and this fact necessitates increased labor on the part of the forces which impel the blood and air whenever an abundant exchange of gases is required.*

Wherever the pneumonia exudation fills the bronchi and alveoli, the exchange of gases can no longer take place. The maximum work of each respiration, whether superficial or deep, is therefore necessarily diminished by the amount normally performed by the portion of lung now diseased. If now a demand arise for the most complete aeration possible, the forces which convey the air and blood to the place where the exchange of gases takes place must be augmented; hence the cardiac and the respiratory muscles must do an increased amount of work. As

before mentioned, the stronger action of the respiratory muscles has again an indirect effect upon the heart.

4. *The fever first brings to expression the local disturbances produced by the pneumonia.*

The fact itself requires no demonstration. Every pneumonia which terminates by crisis, the temperature falling from 3° to 5° within a few hours, shows that the dyspnœa and the more forcible action of the heart are due only to the fever. So complete and rapid a change in the symptoms certainly cannot have resulted from a diminution in the amount of the pulmonary disease. If this evidence be not regarded as convincing, there is still another fact which is not open to attack. The same reduction in the frequency of the pulse and respiration, the same general improvement takes place when the temperature is reduced to the normal by the use of the cold bath, and disappears as soon as the effect of the bath passes off and the temperature rises again. In this case no one would suppose that the local lesions have disappeared and returned again.

Let us now inquire in what way the symptoms in question are produced by the fever.

This question can be readily answered by showing that,

5. *The fever induces increased labor on the part of the heart, and at the same time inflicts a direct injury upon it.*

The most constant symptom of the fever is the elevation of temperature. With the increase of the body heat the pulse becomes more frequent (Liebermeister); that is to say, the number of systoles within a given time increases, and consequently their duration must become longer,[1] and that of the diastoles shorter; in other words, the period of work on the part of the cardiac muscle is lengthened, and its period of repose shortened, and moreover, a greater amount of work than usual is required from it.

When fever is present the formation of carbonic acid is increased (Liebermeister, Leyden). In order to prevent its accumulation in the body, the excretion must be more rapid than normal, and this result can be effected only by increasing the force supplied by the heart and the respiratory muscles in the way which has previously been described *in extenso.*

The fever leads to a degeneration of the muscular substance of the heart (Zenker, Liebermeister). Since the amount of vital force produced by the muscle in its contraction is proportional to its mass, that is, to the number of primitive fibres contained in it, it follows that any diminution in their number is equivalent to a

[1] The theoretically admitted possibility, that as the systoles become more frequent their duration is diminished, is certainly not usually true to any considerable extent. It is only necessary to observe the pulse to be satisfied upon this point.

diminution of their capacity for work. The same thing is true for each primitive fibre. Its capacity for work depends in its turn upon the amount of contractile substance contained in it. Now, inasmuch as it can be demonstrated anatomically that the fever produces not only a diminution in the number of primitive fibres (destruction of a certain number), but also a diminution in the contractile substance of those that are left (accumulation of fat and detritus), it follows that the fever lessens the capacity for work on the part of the heart as a whole. The same result is produced also in the following way: the degenerated part of the contractile substance, in consequence of its inertia and elasticity, resists the contraction of the cardiac muscle, and absorbs a portion of the vital force set free during its contraction. This resistance has to be overcome, otherwise contraction is impossible.

We must also take into the account the indirect harm which is done by the fever to all the organs of the body, as well as to the heart and respiratory muscles. The diminution in the amount of nourishment taken, amounting in highly febrile conditions to almost complete abstinence, and present to a greater or less extent in every febrile state, affects chiefly those parts whose task is the most arduous, the muscles which labor uninterruptedly, such as the heart and respiratory muscles.

From all sides the threads run together to a central point. *It is the heart, and always the heart, upon which the burden is ultimately thrown.*[1] It is therefore the duty of the physician to enable the heart during pneumonia to perform the additional labor made necessary by the disease. This duty involves two subdivisions.

1. *Prophylaxis against exhaustion of the heart.*
2. *Control of already existing exhaustion.*

The fever is the first point of attack for treatment. This is shown by the fact already repeatedly urged, that, notwithstanding the continuance of the local derangement, the force of the disease is usually broken when the fever abates.

In the present condition of our knowledge the prophylaxis against exhaustion of the heart in pneumonia coincides essentially with the treatment of the fever. But there are some points to which I wish to call especial attention in this connection.

On another occasion I laid down a proposition, which I regard as indisputable in the treatment of typhoid fever: *Sine thermometro nulla therapia.* That is to say,

[1] If we examine from this point of view the numerous facts in regard to prognosis which have been confirmed by experience, we shall readily admit that among them all this is the one which dominates the rest.

in this disease the temperature of the body is such a dominant factor, that, with the rare exceptions of intestinal hemorrhage, perforation, etc., the other symptoms are comparatively unimportant. The amount of heat as ascertained by the thermometer is therefore in this case a safe guide for treatment. In pneumonia it is different. Here the proposition should read: *Sine pulsu nulla therapia.* If I were asked whether I would prefer to treat a severe pneumonia without a thermometer or without examining the pulse, I would say unhesitatingly,—without the thermometer. It is necessary to speak positively on this point, because in these times, when we try to measure everything (messlustigen Zeit), we are in danger of losing the *tactus eruditus* of our forefathers. It is all the more necessary, because the thermometer is so simple an instrument that even the most shallow-brained nurse can be taught its use for therapeutical purposes. The critical examination, in fact even the observation of the pulse, requires special skill, which can be acquired only after long practice. The sphygmograph is a valuable instrument, but it is not suitable for clinical purposes except in a hospital. Moreover, if the character of the pulse, and this includes much besides its mere frequency, show that the heart is doing well, the temperature is not to be regarded for the time being as necessarily critical, even if it exceed 104° F. On the other hand, even with an absolutely low temperature, treatment may be required if the heart is becoming too much exhausted.

Let us now consider the means by which the fever is to be combated.

Is it proper to bathe a patient who is ill with pneumonia, to abstract heat directly?

It cannot be denied that in considering this subject we are met by some *à priori* objections which appear very formidable. It may be fairly urged that from the moment when the peripheral vessels contract under the influence of the cold, the bath produces an increased resistance in the vessels, and consequently an increase of work for the heart. It becomes a question, therefore, whether the overloaded heart may not in this way become completely paralyzed. The increased production of heat in the bath, and the increased demand upon the heart and respiratory muscles resulting from the additional excretion of carbonic acid, both point in the same direction.

These are certainly very important considerations. Experience, however, shows that these objections are not very serious. I can safely assert that neither myself nor my pupils have ever met with such a result, although no one, so far as I know, has ever used baths so frequently or so systematically, or at such a

low temperature as myself. I do not deny that fatal collapse may occur during the bath, or that the bath may be a direct cause of death; but I insist that the collapse may be avoided, if we observe a very simple precaution. For the time being it is necessary for the heart to perform additional work. Is it not possible to enable the heart to satisfy this transient demand? Unquestionably; this can always be done. Of course we ought not to experiment upon a patient who is at the last gasp. I say the "last gasp," because if the patient be treated before this time his life can in many instances be saved. The treatment in such cases will be considered subsequently in detail.

Another objection to the use of cold baths, and one which acts as a greater restraint alike upon physicians and the laity in carrying out this plan of treatment, is that patients in this way catch cold. The fear of such a result makes even very intelligent persons as timid as children who have been listening to stories of black men; and with as little reason.

For the sake of releasing this ghost, which has been restlessly wandering about for centuries past, I will here relate my own experience, and explain my views, which I have amply verified, in still another way. A patient with pneumonia requires in the first place fresh air. This should be provided, if possible, without producing a current of air; but if I have to choose between bad air and a draft, I choose the latter, and my patients do well. I allow them even to bathe under these circumstances, and find that the exposure of the naked skin to a draught as they come out of the bath does them no harm. The circumstances of my polyclinic patients frequently compel me to put up with such primitive methods in the presence of a draft from a broken window or an open door, and yet I comparatively rarely see the classical "complications from cold," bronchial catarrh, pleuritis, pericarditis, etc. Nor have I noticed that such sequelæ as chronic pneumonia, phthisis, etc., were apt to occur from this cause. Hence, in case the abstraction of heat were necessary and no water were to be had, I would not hesitate to expose my patient to cold air until the necessary amount of cooling was obtained. This procedure would subject him to much more discomfort than would result from the water-bath, but I am

certain it would do him good. In opposition to the scruples on this point, which are theoretically well founded, and to the mere prejudice which has no foundation at all, I think I am justified by my experience in saying that the direct abstraction of heat in pneumonia is proper treatment, and I am happy to say that I am supported by the weighty authority of Liebermeister.

DEATHS FROM PNEUMONIA UNDER ANTIPYRETIC TREATMENT:

The Basle Clinique

Classes by Age.	Sex.		Remarks.
	Male.	Female.	
15–20 years	2	..	(1.) 17 years. Tuberculosis. (2.) 19 years. Phthisis.
21–30 years	2	..	(3.) 25 years. Complication with pleuritis. (4.) Complic. with degener. cordis.
31–40 years	8	..	(5.) 32 years. Complic. with pericarditis. (6.) 33 years. Morbus Brightii. (7.) 34 years. Intemperate. (8.) 35 years. Hæmoptysis, gangrene. (9.) 36 years. Pleuritis, died day after reception. (10.) 36 years. Intemperate. (11.) 38 years. Emphysema. (12.) 40 years. Degenerat. cordis, intemperate, meningitis.
41–50 years	1	7	(13.) 47 years. Male. Intemperate. (14 and 15.) Females. 41 and 42 years. Without complications. (16.) 41 years. Female. Pregnancy, pleuritis. (17.) 43 years. Female. Phthisis. (18.) 45 years. Female. Gangrene. (19.) 45 years. Female. Pregnancy, meningitis. (20.) 49 years. Female. Struma.
51–60 years	3	4	(21.) 51 years. Intemperate. (22 and 23.) Males. 56 and 59 years. Without complication. (24.) 60 years. Female. No complication. (25.) 52 years. Female. Degener. cordis. (26.) 53 years. Female. Pleuritis. (27.) 57 years. Female. Pericarditis.
61–70 years	3	5	(28.) 64 years. Male. Intemperate, degener. cordis. (29.) The same. (30.) 67 years. Male. Degener. cordis. (31 and 32.) Females. 62 and 65 years. Without complications. (33.) 62 years. Female. Caries, emphysema. (34.) 66 years. Female. Cerebro-spinal meningitis. (35.) 70 years. Female, degener. cordis.
71–80 years	3	..	(36 to 38.) Males, 70, 72, 73 years.

This tabular compilation, which I have made from the fully detailed records of the autopsies, shows that only ten of the fatal cases were uncomplicated, and that all

of these, with the exception of two women, were over fifty-five years of age. This satisfactorily explains the high percentage of mortality notwithstanding the antipyretic treatment.[1]

Compared with each other the two methods of treatment show the following contrast:

From 1839–1866. The usual routine treatment.
652 patients; mortality, 25.2 per cent.

From 1867–1871. Antipyretic treatment.
230 patients; mortality, 16.5 per cent.

Not satisfied with this result, which might be questioned, Fismer compares 230 carefully observed cases of pneumonia occurring between 1858 and 1866 with the 230 occurring during the period in which the antipyretic treatment was used. The conditions which exert a decisive influence upon the mortality must be much the same in both of these classes of cases, so that any difference in the result should be ascribed to the treatment.

1858–1866. No antipyretic treatment
230 cases; 172 males; 58 females.
60 deaths; 41 males; 19 females.
26.1 per cent.; 23.8 per cent.; 32.8 per cent.

1867–1871. Antipyretic treatment.
230 cases; 166 males; 64 females.
38 deaths; 22 males; 16 females.
16.5 per cent.; 13.3 per cent.; 25 per cent.

The reduction in the mortality is 9.6 per cent. The total mortality is still very high, but may be explained by local causes.

In regard to the amount of heat to be withdrawn, I again take as my guide the principle that the abstraction of heat is intended to guard against exhaustion of the heart, and that the chief danger of such exhaustion lies in the fever.

In strong persons, who are only moderately ill, and have no complications, we may follow the rule usually adopted in the hydropathic treatment of typhoid fever. A bath at the temperature of well-water should be administered as often as the temperature in the rectum reaches 104° F. The duration of the bath

[1] In his much-quoted and standard work, Fismer has given a careful analysis of the results of treatment at the Basle Hospital. The fundamental principle of the treatment was the reduction of the fever. This indication was carried out chiefly through the abstraction of heat by means of baths of different temperatures. In addition to this, antipyretic remedies were used, such as quinine, digitalis, and veratrine. Digitalis was prescribed only with great caution. Baths were used whenever the temperature in the axilla was above 102.2° F.

should depend upon the effect obtained, and should vary from seven to twenty-five minutes. This simple rule answers for all the different indications arising from the size of the patient, the amount of fat, the intensity of the fever, and the temperature of the bath. But it does not apply to the treatment of all cases of pneumonia. Every one knows that cases often occur, especially among the aged or very fat persons, or those of feeble constitution, in whom the temperature never, or at least only rarely reaches 104° F., but varies between 101° and 103°. These cases require special care. In such persons I prefer to take advantage of the fact that in all the ordinary febrile diseases the rise and fall of the temperature are distributed over the twenty-four hours in the same way as in health. Tepid baths (77° to 78° F.) administered for twenty or thirty minutes at a time early in the morning (four to seven o'clock) will reduce the temperature in these cases for a comparatively long period. The effect may be continued by giving quinine at the same time. As long as the low temperature lasts, the heart, so to speak, takes a rest. Of course the baths may be repeated at any other hour in the day. The careful examination of the pulse will readily detect when the proper effect of the bath has been attained.

With young children the wet sheet may be used. The application of cold linen cloths around the chest only is not sufficient, because the amount of heat abstracted in this way is too small for any but children under one year of age. At the same time the thermometer must be repeatedly consulted. I do not quite like these packings, because they cause much more trouble and discomfort to the attendants and patient than baths, and are much less certain in their effect.

On the other hand, there are cases of pneumonia in which the violence of the fever, as shown not merely by the absolute height of the temperatures, but also by the resistance to the abstraction of heat, can be broken only by the most energetic and rapidly repeated efforts to lower the temperature. In such cases we have no choice; we must either let the fever run its natural course, and then the patient generally dies, or we must not be afraid of exerting our authority and carrying out to their ultimate results the opinions which we believe to be correct. By so

doing, we of course expose ourselves to the danger of being regarded by the public as responsible for the result, if the case terminate unfavorably. No conscientious physician will hesitate which course to adopt, if he is satisfied of the truth of his opinions.

Several years ago I was much impressed by the extent to which these baths can be used, as shown in the case of my daughter, then only nineteen months old, who for the third time within a brief period was attacked with a severe pneumonia. The temperature rose above 105.8° F., and returned so quickly after baths of 60.8° F. that I found myself compelled to reduce the temperature of the water to 41° and 42.8°, and to continue them for ten minutes. My child recovered, and at no time during the employment of these extreme measures, extending over several days, was there the slightest indication of collapse. Since that time I have repeatedly treated patients in this manner without bad results. One precaution, however, I must most positively insist upon; a patient with pneumonia must never be bathed without the administration of stimulants before and afterwards, and the amount of stimulant must be increased when water of a lower temperature is used, or the duration of the bath is lengthened. I have previously explained why the heart should be somewhat stimulated before the bath. Stimulation is necessary also after the bath is finished, because the cooling process, which does not reach its maximum until fifteen minutes or half an hour later, is very apt to produce symptoms of collapse in persons with feeble hearts. Moreover, it seems to me that such stimulation effects a more rapid distribution of the heat throughout the whole body.

In cases of moderate severity, such as I have already described, I usually give one or two tablespoonfuls of red wine before and after the bath; but, whenever there is the slightest indication that the heart is not performing its work satisfactorily, I use port wine or Madeira, or perhaps even champagne. If the bath be quite cold, I give from one to three large tablespoonfuls of wine about five minutes before the patient enters the bath, and I repeat the quantity while he is in it, and immediately after he leaves it. Under such circum-

stances the wine must always be one of the stronger kinds. In children the quantity should be proportionately less; but when the attack is severe I give a relatively larger amount to children than to adults. No absolute rules can be laid down; the quantity must be determined by the character of the pulse. Once more let me insist most positively, that if we wish to treat pneumonia by cold baths without unfortunate accidents, we must not spare stimulants.

The indications for the treatment of pneumonia by warm bathing are of the same character. In such cases the heart is generally in a weak condition from the start, and less capable of work, and we must expect a more protracted course of the disease.

It would be very injudicious to commence at the outset with the stronger stimulants; but if we are compelled to resort to cold bathing, they will of course be indispensable. A little experience will enable us to find our way with safety.

In addition to the direct abstraction of heat, I always use quinine. Above all other antipyretic medicines it possesses the invaluable advantage of reducing the temperature without injuring the heart, and this it accomplishes by diminishing the production of heat.

Tartar emetic and its younger brother veratrine diminish the temperature only at the expense of the heart; both induce collapse. When digitalis is used the effect upon the heart takes place very late. We fail, therefore, to obtain its control in regulating the action of the heart at the very time when we require it. Has not every one who is in the habit of prescribing efficient doses of this remedy seen cases in which the collapse induced by the digitalis coincided with that of the defervescence? I venture to say that any one who has ever seen such a result is not particularly anxious to repeat the experiment. The use of digitalis as a regulator of the heart's action in pneumonia is a very different matter. In cardiac insufficiency produced by muscular degeneration, with or without valvular disease, it may be absolutely necessary to give digitalis, notwithstanding the existence of croupous pneumonia. I have repeatedly had to do this, especially in old persons. But here we should use the infusion of digitalis in small, not too frequently repeated doses. Given in such quantities we avoid its paralyzing influence upon the heart, but at the same time we lose its effect upon the temperature. I cannot understand the object of using digitalis in pneumonia only for the purpose of reducing the frequency of the pulse from 100 to 80 beats, an effect which it requires doses of some size to produce. It was not such a control of the heart's action that I had in view

when I recommended the contingent use of the remedy. At the same time I am willing to admit that when the heart's action is vigorous, the drug may in many cases not only be borne without harm, but may be used with advantage to reduce the temperature.

But are we justified in using a remedy which may perhaps seriously injure the patient, when a greater effect can be produced in a way which is certainly unattended by danger?

The antifebrile action of venesection is slight and uncertain. The physician who bleeds in pneumonia on account of the fever, resembles the philosopher who cuts down the fruit-tree in order to get the fruit. The advocates of this indication for bloodletting furnish in their own reports of cases the most convincing evidence for the inadmissibleness of the measure. At all events, this indication should disappear from the text-books. To the conscience of the weak man, whom fate makes a physician as a punishment to his fellows, it serves as a welcome salve, when he lets blood merely to gratify a popular demand and establish his own position.

When properly used, quinine diminishes the temperature for at least twelve hours. The greatest reduction (2.7° to 4.5° F.) takes place from five to seven hours after the medicine is taken; the line of descent and subsequent elevation is very nearly straight, as I have demonstrated by thermometrical measurements repeated every five minutes.

There is much to be said in regard to the mode of administering it. The formula below is the one I use as a suitable dose in moderately severe pneumonia in an adult:—

℞ Quiniæ sulphatis ..... xxx.
Acidi muriatici ..... q. s.
Aq. destil. ..... ʒ iiss.
Misce. To be taken as one dose.

This should be given in the evening between six and eight o'clock. For children I use a grain and a half for every year up to five years of age, and after that period from seven to fifteen grains, according to circumstances. These quantities may be exceeded without doing any harm. When the fever is intense, seventy-seven grains may be given to a strong adult, and fifteen grains to a child under one year, always in one dose. I have repeatedly used both of these amounts. I have acquired my experience by gradually increasing my doses, and I have never seen any harm done; in fact, it is my firm belief that even these are not the extreme limits as to quantity. I know that many will be alarmed at such large doses. My teacher is experience. Only fools resist facts. The physician who regards it as his duty to cure the sick, does not treat his patients according to traditions, but he knows what he wants and is never afraid to do it. I have only one caution to give. In those cases in which the temperature has at some time been 105.8° F., and has risen again rapidly after

an unsatisfactory abstraction of heat, it is not necessary to give forthwith seventy-seven grains of quinine; this should be done only when the repeated use of decidedly cold baths has lowered the temperature for but a short time, and smaller doses have proved useless. In such cases I have first tried the effect of a dose of forty-five or sixty grains. Once more I insist it is the heart which is the guide for the physician. Whoever carefully examines the pulse walks safely; whoever fails to do it, easily stumbles. To object to the use of quinine because "Binz says it is a cardiac poison," savors too much of naïveté as well as lack of experience. Such domestic remedies as black stockings, or covering the feet with lamp-black as a substitute for the general bath, as recommended by Paul Niemeyer, the quinine-hater (Betz, Memorabilien, Jahrg. 1873, 3. Heft), I have never tried.

In my cases I have never noticed any harm done by quinine. The pulse falls at the same time with the temperature, but remains full and strong, or is improved in character if it had previously been weak. The discomfort on the part of the patient is generally not much greater from large than from small doses, and disappears rapidly. Not very infrequently vomiting occurs soon after the quinine is taken. If the medicine have been retained as long as half or three-quarters of an hour, it is scarcely necessary to repeat the dose, because the salt, when exhibited in the form suggested, is rapidly absorbed. If delay be dangerous, the dose may be repeated immediately, because it is better to give too much than too little.

But in many cases the medicine is rejected within a few minutes after it is taken. This may often be prevented by directing the patient to bend forwards as soon as he has swallowed the quinine, and to keep his mouth open so as to allow the abundant flow of saliva, which is usually excited, to run out. Small pieces of ice, swallowed when nausea is first felt, will often prevent the vomiting. If it occur notwithstanding, I usually give a second dose a quarter or half hour afterwards, and perhaps even a third dose. Usually the second one is retained.

If we fail, as we sometimes will, to introduce the quinine into the body by this mode, we may administer it by enema. This plan is almost always successful; absorption seems to take place nearly as quickly by the rectum as by the stomach. If the quinine is to be used by injection, the apothecary should be directed to add but a small amount of acid, and to use three or four times more water for the solution than I have recommended. If much acid be used it irritates the mucous membrane of the rectum excessively, and soon induces the expulsion of the clyster. The use of a mucilaginous vehicle and a few drops of laudanum will be sufficient, with rare exceptions, to produce a sufficient tolerance on the part of the intestine.

In order to get the full effect of each dose of quinine, the latter should not be given daily but every second evening, thus allowing forty-eight hours to elapse between two doses. We can, of course, by unnecessarily frequent doses produce a daily reduction of temperature, but the reduction will not be so great as in the other mode of administration.

Our views as to diet will be influenced by our position upon the leading question, should we feed fevers? For myself, I have long since come to a conclusion upon this point. The idea that nourishment increases the temperature, and should be withheld because we ought not to pour oil upon the fire, seems to me untenable, even if we were in possession of evidence that the supposed elevation of temperature actually took place, which cannot be said to be the case. But even if it were, I would still feed my fever patients, for the nourishment supplied to the body protects its tissues from greater loss and renews what has already been lost. If the additional heat generated by the oxidation of the food be sufficient to raise the temperature too high, we can get rid of the dangerous excess by cold baths. In a total abstinence from nourishment, a portion of the formed tissues is necessarily sacrificed to satisfy the eager demands of the oxygen in the blood, and the body thus suffers an unnecessary loss. Experience shows that fever patients who are properly fed recover much more rapidly than others, and the reason for this seems to me sufficiently obvious.

The only question, therefore, is to find the proper kind of nourishment. This we shall be able to do the more readily, if by proper treatment we prevent the temperature from remaining too high; we can then expect far more from the organs of digestion. The regularly repeated cleansing of the mouth and teeth is, in my opinion, an important aid in maintaining the appetite. The same purpose is fulfilled also by seasoning the food with a sufficient quantity of common salt, which is valuable, too, on account of its nutritious properties.

If the patient will take food at all, I give him, several times a day, some rare finely scraped meat with bread and butter; the best plan is to give it in small portions three or four times a day, about an hour after a bath. I never force it upon a patient. In cases which are at all severe I insist also upon the patient's taking, in one or two tablespoonful doses, a strong bouillon with one or two eggs daily. Also in the course of the day milk should be given in quantities suitable to the case. When there is dyspeptic derangement the milk should be thinned with from one to three parts of water, and common salt added in sufficient quantity.

In the antipyretic treatment of pneumonia I regard it as a *conditio sine qua non* that the patient should take light wine in amount suitable to his age and habits; for an adult say from half to a whole bottle daily. The portion which is not used just before and after the bath may be mixed with water and drank

at pleasure during the day. I have no objection also to a good beer. There is no doubt that alcoholic drinks lower rather than elevate the temperature, and it is more than probable that the alcohol acts as a direct preservative of the tissues. The former prejudice, which is still prevalent, has been shown by Bouvier and Binz to be unfounded.

There are two other points to be considered which are of considerable importance in regard to the prophylaxis against exhaustion of the heart: I refer to pain and sleeplessness, symptoms which should not be allowed to go untreated.

It is certain that continuous pain in every way diminishes the power of resistance to disease. Moreover, the pain resulting from the inflammation of the pleura interferes with respiration. In these cases I have found local hypodermic injections of morphine very useful. The dose may be small; from one-sixth to one-fourth of a grain is generally sufficient. The same remarks will apply to the distressing cough which is frequently present. If the pain be excessive, the treatment for its relief will for the most part coincide with that for the induction of sleep. In no case of pneumonia should insomnia be permitted to continue. Probably every one has at some time had occasion to contrast his physical condition after a night of watching with that after a night's sleep. Such a deprivation of rest affects a person already weakened much more than a strong one, and I therefore regard it as an important indication for treatment in pneumonia. In many cases, however, the insomnia disappears without farther treatment as soon as antipyretic measures are adopted. Should this not be the case, I use narcotics in doses sufficiently large to produce the desired effect. In children the antipyretic treatment will, with very rare exceptions, be sufficient.

There are some cases in which the whole treatment must for a time be directed to the production of sleep. Besides complete insomnia there are also continuous delirium and jactitation, and frequently actual maniacal attacks. Sometimes, when the antipyretic treatment is ineffectual, a full dose of quinine answers the purpose; in many febrile conditions this drug seems to act directly as a hypnotic. To this class belong also those cases of pneumonia in drunkards, which are characterized by the frequent occurrence of delirium, and yet lack some of the classical symptoms of delirium tremens. Here also may be classed delirium tremens occurring in connection with pneumonia.

Whatever may have been the original cause of the feebleness of the heart, the incessant muscular action in these cases throws upon that organ a constantly increas-

ing amount of work. The pulse very soon shows that the heart is unequal to this task. Sleep, and the cessation of muscular activity associated with it, is the only remedy. In such cases I have always produced sleep by very large doses of a suitable remedy. Of late I have generally used the hydrate of chloral in a single dose of from seventy-seven grains to two drachms, if smaller quantities fail. In febrile cases we should never forget to give either before or after the administration of this drug a sufficient quantity of properly diluted hydrochloric acid, in order to prevent a decomposition of the chloral hydrate upon the feebly acid or actually alkaline mucous membrane of the stomach. Nor, whatever remedies we employ, should we forget the active use of cardiac stimulants. Without this precaution energetic medication should not be adopted; with it, we need not fear disobeying the warnings of the pharmacopœia, provided it be indispensable that sleep should be induced.[1]

In all severe acute diseases I disapprove of darkened bedrooms. It is sufficient to place the bed in such a position that the light does not shine directly into the eyes of the patient. In my opinion patients who are exposed to the light make the best recovery.

We come now to the second part of our subject, the *treatment of already existing exhaustion of the heart.* In those cases in which the patient has been under treatment from the start, and in which prophylactic measures have proved insufficient, we should bear in mind the rule, that the earlier the approach of cardiac exhaustion is detected, the more easily shall we succeed in overcoming it. The reason for this is obvious. The weakness of the cardiac muscles produces a retardation of the circulation, which leads to a derangement of nutrition in all the organs, and the longer this retardation continues the more profound are the disturbances induced by it. If now by increasing the activity of the heart the normal rapidity of the circulation be after a time restored, there will still elapse a certain interval corresponding to the duration of the derangement, before the mischief is completely removed. This mischief will be most apparent in those parts in which the demand for blood is greatest, that is, in the cardiac and respiratory muscles which are constantly at work. I regard this as a very important fact, and I am constantly on the watch, therefore, for the first symp-

[1] See my article, "Ueber die Behandlung des Säuferwahnsinns." Deutsche Klinik, 1872, No. 11.

toms which indicate a decline in the activity of the heart. I think it is to this watchfulness that I am to a considerable extent indebted for my clinical results. Besides, the condition of the pulse, the ratio between the pulse and the respiration, and the increased cyanosis, which very soon makes its appearance, should be carefully watched. In the early stages of collapse the temperature remains about the same.

In many instances these symptoms are only sufficient to excite suspicion; I am inclined to think that these slight and very trifling disturbances which a careful examination will detect in almost every case, are generally connected with a further spread of the local disease, which for a long time may not be indicated by the thermometer. In such a case it must be left to the judgment of the physician whether he will treat them or not. If the patient be vigorous, I do not usually treat them until they become more pronounced, but I am sure that early treatment does no harm.

The main symptoms of strongly marked collapse, accompanied by œdema of the lungs, are so characteristic that they stamp themselves indelibly upon the memory of every one who has ever seen them. When they occur it is apparent to the dullest eye that they are the beginning of the end. A considerable amount of collapse is almost certainly fatal, unless arrested by treatment. Whatever is done must be done quickly and effectively. There is much difference of opinion upon what the proper treatment is in these cases; and as the subject is a very important one I shall discuss it thoroughly.

In my exposition thus far I have continually spoken of exhaustion of the heart, but I have never mentioned how its results are manifested.

It has already been shown that in pneumonia the chief burden is thrown upon the right ventricle. If the heart fail, the failure shows itself first in this part. Passive congestion occurs in the pulmonary circulation, the left ventricle becomes empty, and in cases of serious failure the blood accumulates in the veins of the general circulation in consequence of the obstruction to the outflow from the imperfectly emptied right ventricle and the diminished *vis a tergo*. Under these circumstances œdema of

the lungs may also occur; and in fatal cases this condition is probably always present. The œdema of the brain, in regard to which there has been much discussion by writers, may be induced by passive congestion in the district of the superior cava; but this is a very rare occurrence. The symptoms described as due to cerebral œdema may, however, be regarded as of the same nature as those produced by a continued fever in connection with an imperfect exchange of gases, or an insufficient blood respiration. Similar symptoms occur in all severe febrile affections, and just as infrequently as in pneumonia. The imperfect filling of the left ventricle produces, moreover, always and under all circumstances very injurious results. The working muscles, the cardiac and respiratory, do not receive a sufficient supply of blood, and consequently their capacity for work is seriously threatened. It is, in fact, more correct, therefore, to ascribe the fatal result to the insufficiency of the heart rather than to the pulmonary œdema.

This is something more than a change of terms. When we speak of œdema of the lungs we are far too apt to imagine that the only difficulty is that the air and blood are separated from each other mechanically by the serum effused into the alveoli, thus interfering with the excretion of carbonic acid and producing its fatal retention in the blood. There is no doubt that the exchange of gases is rendered difficult by the filling of the alveoli with fluid; but, besides this, another circumstance is to be taken into account, viz., that in consequence of the retardation of the circulation, which precedes the occurrence of the œdema, and is caused by the feeble action of the heart, the exchange of gases is delayed everywhere in the *tissues*, with retention there of all the products of oxidation. It is better, therefore, to ascribe the fatal result to the insufficiency of the heart, which is the cause, and not to the œdema, which is merely an effect.

Within the last ten years a view has been advanced which is totally at variance with the one just given. Felix Niemeyer[1] refers so constantly to a pulmonary œdema in pneumonia, resulting from collateral fluxion, that the reader, if he be unacquainted with the subject, is persuaded to believe that this is a common occurrence.

The proof of his opinion is briefly this: The closure of a portion of the efferent

[1] Handbuch, l. c.

vessels in the pulmonary circulation by the pneumonic exudation causes a fluxion, an increased and quickened afflux to the other vessels. Now, the capillaries in the lungs are not, as in other organs, "embedded in a more or less resistant tissue, but lie, with a large part of their walls almost or quite naked, in a cavity containing air, and air, moreover, which becomes rarefied in each inspiration." They are consequently unable to withstand the increase of blood pressure. They first become distended, and then their contents escape.[1]

Niemeyer is led by these considerations to believe that stimulation of the heart must increase the amount of œdema already existing in the alveoli. That this influence is incorrect can be easily shown—

1. If these gross mechanical conditions be the controlling factors, why do we not always find collateral œdema whenever the disease attacks a robust individual? Why do we see pulmonary œdema in the feeble, and fail to find it in the strong?

2. In emphysema of the lungs, which is treated by Niemeyer himself strictly from the same point of view—viz., diminution in the number of capillaries—the right ventricle is always hypertrophied, its contractions are consequently more forcible, and in the pulmonary capillaries, which are distended by the "collateral fluxion," the pressure is increased. Œdema of the lungs does not occur, however, so long as the right ventricle is hypertrophied, but only when the muscular fibres exhibit a fatty degeneration. How can this fact be reconciled with the above theory?

3. During œdema of the lungs we never find an increased intensity of the sounds over the right ventricle or pulmonary artery (Thierfelder).

4. I have seen in all probability a thousand cases of pneumonia. Among these, as nearly as I can distinctly recollect, there was one patient who on two occasions had œdema of the lungs twenty-four hours after the invasion of the disease. In all the other cases the œdema occurred later. This one was treated by very active stimulation and recovered. I cannot give any positive statistics, but I think I can probably trust my memory, because for some years I have directed my attention to this point.

I remember very distinctly two cases of pneumothorax in which a few hours after its occurrence paralysis of the heart and œdema of the lungs took place. In these cases also, where the collapse of a whole lung afforded the most favorable opportunity for the occurrence of a "collateral active œdema," the latter disappeared after the employment of the strongest stimulants administered in rapid succession. The experience of a single individual is only a drop in the ocean; but how many physicians do see œdema of the lungs in pneumonia during the period of active local changes? How many of us see œdema of the lungs in pneumonia, along with increased activity of the heart's action? I think that I may be allowed to dismiss this subject without further discussion. Even if it cannot be shown that it is theoretically impossible for œdema to arise in this way, still experience teaches that its

[1] Niemeyer goes still farther; he ascribes the hyperæmia and œdema in the lungs solely to the increased action of the heart.

occurrence is exceedingly rare. When it does occur, there is probably present a special physiological as well as special physical conditions, viz., a diminution in the resistance of the vessels, as in persons disposed to phthisis, in drunkards, etc. If the purely mechanical conditions insisted upon by Niemeyer were really the effective causes, the œdema of the lungs would necessarily occur more frequently at the very outset of pneumonia.

If in consequence of a temporary inability of the right heart to overcome the resistance in the pulmonary circulation, passive œdema take place, what should be done? The routinists teach that a diminution in the quantity of blood in the body lessens the work to be performed by the heart, because the mass to be moved is smaller. At the same time, in consequence of the absorption which takes place on the part of the tissues and the surface of the internal organs, the effused fluid in the pulmonary alveoli disappears. Venesection also lessens the force of the heart's action and permits the absorption of the pulmonary œdema to take place. Venesection is therefore clearly indicated in passive pulmonary œdema. Experience shows that under these circumstances the most alarming symptoms do, as a matter of fact, yield to bloodletting, and often very rapidly, so that there are few measures which can produce a more striking result.

And yet, is this remedy a really judicious one, or have we others by which the same effect can be accomplished? If bloodletting were an indifferent measure, discussion would be unnecessary; but it is probably the least indifferent of all measures. What is the usual result when venesection is practised on account of œdema of the lungs in pneumonia? Twenty-four hours afterwards—often much sooner—the condition is the same as before the venesection. Again the lancet is used, and again the symptoms yield. If the natural termination by crisis do not occur very soon, a third and fourth bleeding become necessary, and the patient certainly dies. Bloodletting should not be resorted to even on a single occasion except for the purpose of gaining time for the occurrence of the spontaneous termination of the disease. If there were no other way of accomplishing this result I would employ bloodletting in a given case in order to gain time. But cannot the same thing be effected in a less

dangerous way? If we can succeed in stimulating the heart to increased labor until the obstacle in the pulmonary circulation is overcome, the problem will be solved. Experience must then decide the question whether this increased activity may not be quickly followed by so much exhaustion as to make this mode of treatment as injurious as bloodletting.

Let us consider the chief points of these two methods.

Every laboring muscle requires oxygen; the more oxygen the more vigorous its action. *Ceteris paribus*, the amount of oxygen supplied to the muscles is dependent upon the number of red blood-corpuscles. These are diminished by bloodletting. Less oxygen will therefore be taken up by the blood after venesection. If the amount of oxygen in the blood be insufficient for the demands of the system, the blood must circulate more rapidly in order to supply the demand. Because, as the number of red blood-corpuscles remains the same, the only possible way in which the demand can be met is by increasing the surface of contact between the blood and the air in the lungs for absorption, and between the blood and the tissues for excretion. Furthermore, the relief to the heart by bloodletting is very transient, because the volume of the blood is rapidly restored through absorption by the tissues. To state the case in a single proposition: the cardiac and respiratory muscles are compelled to do more work after bloodletting than before, in order to convey to the tissues the same amount of oxygen.

The demand for oxygen by the body is, moreover, diminished by bloodletting only when the temperature is considerably reduced by it, and even then only so long as the reduction continues. As a rule, the demand for oxygen after venesection is the same as, or even greater than, before; and this explains the very short duration of the improvement from this remedy. It is the lessened demand for oxygen, the crisis, which first produces a diminution in the labor of the internal muscles. Bloodletting is a very doubtful mode of escape from the difficulty. It may be compared to a usurer who is willing to keep on advancing money on good security, in order to finally get possession of all his debtor's property. For the time being the loan relieves the

embarrassment, but unless the expenditure lessens, the catastrophe is only a question of time.

I must refer to one objection, which is, however, more plausible than real. If the above conclusions be correct, it is urged, how does it happen that immediately after bloodletting the heart and respiratory muscles act more vigorously and effectively than before? The explanation lies in the fact that the burden is momentarily so far removed from the right side of the heart, that it is enabled to impel more blood into the left side. But if the left ventricle receive more blood after venesection than before, it follows that the heart and respiratory muscles will also receive more oxygen in the excess of blood conveyed to them.

Stimulants act very differently. They not only spur the cardiac muscle to perform absolutely more work, but they also directly enable the heart to perform it. Every vigorous pulsation of the heart forces more blood out of the overfilled right ventricle into the left, and benefits first of all the heart itself by supplying it with more oxygen, and removing the accumulated débris of oxidation. The quantity of blood is left unchanged; it is only necessary to call forth a temporary increase of exertion in order to relieve a momentary embarrassment. Under these circumstances the stimulant is in fact not only a whip to the heart, but oats besides, because it supplies the organ with oxygen, without which muscular work is impossible.

After this theoretical raisonnement our decision cannot be doubtful. If this road be passable, it is the one to be chosen, and that it is passable has been demonstrated by experience.

It is possible by the proper and bold use of stimulants to maintain life in pneumonia for at least three or four days after the heart has shown indications of exhaustion, and very often for even a longer period.

In the less serious forms of cardiac exhaustion, a full dose, say four ounces, of a strong wine, such as port, Madeira, sherry, etc., will be sufficient in the majority of cases to relieve it. The size of each dose and the frequency of its administration will of course depend upon the individuality of the patient, especially upon his habits in regard to wine. If these milder attacks recur frequently, I prefer to use an emulsion of camphor (two scruples to six and a half ounces of water, a tablespoonful every two hours). If the symptoms continue, without, however, becoming

at any time alarming, I order every hour or half hour a tablespoonful of strong wine and camphor emulsion alternately. Should sudden and severe collapse take place, it is proper to give musk (from three-quarters of a grain to two grains at a dose) with one or more tablespoonfuls of champagne, given at intervals of from ten minutes to half an hour until improvement occurs. Musk acts rapidly like champagne, camphor more slowly, but its effects last longer. In severe cases, with more frequent intercurrent attacks, I prefer therefore to continue the camphor after the musk and champagne, in order to maintain the desired effect. When the patient is no longer able to swallow, and cannot retain an enema, I have of late frequently used a hypodermic injection of camphor. The simplest plan is to take the camphorated oil of the pharmacopœia[1] (one part of camphor to nine of olive oil), and to inject by means of a Pravaz syringe as much as seems to be necessary. The effect is rapid. Abscesses do not result from the injection. Even more rapid still in its effects than musk and champagne, is hot grog; from one to two parts of cognac or rum, or whiskey if nothing else is to be had, to one part of water, or perhaps one part of strong coffee or tea. Of this I give one or more tablespoonfuls every ten minutes. In many cases the effect is wonderful, but it does not last long and requires to be kept up.

To lay down a set of rules for the administration of stimulants would be a very thankless task. Let the principles of treatment be mastered, and then quiet observation at the bedside will soon give one the experience which inspires confidence. A timely attention to the therapeutics of cardiac symptoms generally makes the use of heavy artillery unnecessary, but if we are obliged to bring this into the field, it should be borne in mind that it is unnecessary to place any limit to the doses of stimulants; if the weaker stimulants fail, we may use the stronger and increase the dose. In such cases the only limit is consistency; whoever is timid where this is at stake, really belongs elsewhere than at the bedside.

The advocates of bloodletting have, moreover, in fact advanced so far that they now use stimulants after the operation. That within certain limits something may be accomplished in this way may be conceded; but I am not afraid to go the whole distance, and treat œdema of the lungs from the start without bloodletting. The first attempt will not be the last.

Is it advisable or necessary to bathe the patient as soon as the symptoms of cardiac exhaustion have become distinctly recognized? Theoretically we reply: The bath increases for the time being the work of the heart, but diminishes on the whole the amount of work to be performed by it. If the heart can bear the temporary increase of labor without danger, then baths may be used, otherwise not. Practically, the matter stands

[1] *I.e.*, the German Pharmacopœia: the U.S. and British officinal "liniments of camphor" are rather more than twice as strong.

thus: Unless the collapse be extreme, most patients can be treated by the direct abstraction of heat very satisfactorily, if sufficient care be used in the choice of stimulants and the temperature of the baths. Quinine in large doses is tolerated in all cases. My experience has gradually led me to give larger and larger doses. Any one who has been accustomed to treat pneumonia in accordance with these principles, and has had a low percentage of mortality, will generally act with more boldness than a novice in such treatment. *It must never be forgotten that the most dangerous enemy to the heart is the high temperature, and that this may be safely and quickly lowered by bathing.*

There is one other fact to which I wish to call attention. Not infrequently in exhausted patients collapse will suddenly occur at the time of the crisis or even a few days afterwards. These symptoms generally very soon disappear spontaneously, but sometimes they are dangerous. They are less frequently met with, in my opinion, if the use of a light wine be continued for five or six days after the defervescence. The attendants upon the patient should be prepared for the possibility of such an occurrence, and they should receive instructions beforehand, because much depends upon timely treatment. I seek to promote the convalescence of my cases by giving the most abundant nourishment, especially albuminates, and by caution in the use of much beer or wine. I am also partial to the employment of ferruginous preparations, especially the following:

℞ Ferri redacti..... ʒ ij.
Extracti cinchonæ..... gr. xxx.
Cinnamomi..... q. s.
Misce et divide in pil. no. 100.
S. Three pills three times daily, ten minutes after eating.

If there be much dyspepsia, begin with one pill and increase gradually.

The view here presented has this advantage from the start, that it develops the whole theory of the danger in pneumonia from a single point of view, and also clearly indicates the proper treatment. But in therapeutics the result is our only guide, and every deduction, however attractive it may appear, is worth-

less unless based upon clinical experience. I am conscious that my views have not been formed at the writing-desk, but at the bedside, and that my theory springs from practice.

However, by their fruits ye shall know them. I have no statistics to present, but only a simple compilation of 200 cases which were treated in the Kiel polyclinic, and 48 in that of Tübingen, according to the principles which I have described. No selection has been made, but the cases are taken in the chronological order in which they were recorded. A statistical report of pneumonia, which can claim any value, must, I take it, be merely a record of deaths. Otherwise there is too much room for chance, unless one has thousands of cases to offer. The examination of the tables will show that the treatment has effected all that could be reasonably expected from it. I regard it as proper to include every case of death which was diagnosticated clinically and at the autopsy as "pneumonia." This excludes all arbitrariness. Moreover, when I compare 400 cases treated in this way with 400 cases equally unselected, which were treated at the polyclinic before my principles were carried out, it appears that the mortality has been diminished just about one-half. But even these figures are too small for me. Let my record of deaths speak for me.

KIEL POLYCLINIC.

Classes by Age.	Sex. Male.	Sex. Female.	Deaths.	Remarks.
Up to 1 year	6	3	0	
	4.5 per cent.			
1–5 years	34	29	2 [1]	[1] *a.* Boy, 1¼ years old; terminal pneumonia in follicular catarrh of the intestines. *b.* Girl, 1¼ years; terminal pneumonia in multiple circumscribed gangrene of skin. Lived in a most horrible cellar.
	31.5 per cent.			
6–10 years	20	18	2 [2]	[2] *a.* Girl, 7 years; miliary tuberculosis after pertussis, with chief localization in the meninges; finally pneumonia also. *b.* Boy, 6 years; pericarditis as complication of pneumonia on left side, involving the linguiform process. Purulent effusion removed by operation. Death from cerebral meningitis.
	19.0 per cent.			
11–20 years	10	6	0	
	8.0 per cent.			
21–30 years	5	8	0	
	6.5 per cent.			
31–40 years	11	3	2 [3]	[3] *a.* Female, 33 years; abortion with uncontrollable hemorrhage; death with symptoms of acute anæmia. *b.* Male, 33 years; terminal pneumonia in diabetes mellitus.
	7.0 per cent.			
41–50 years	11	4	8 [4]	[4] *a* and *b.* Delirium tremens; males, 46 and 50 years. *c.* Male, 49 years; extreme marasmus of drunkards. *d.* Female, 42 years; abortion with profuse hemorrhages. *e.* Female, 46 years; strongly marked marasmus; (vagrant). *f.* Female, 45 years; acute rheumatism, pericarditis, complete adhesions of both pleuræ. *g.* Male, 46 years; epidemic cerebro-spinal meningitis. *h.* Male, 47 years; died a few hours after admission; autopsy: purulent liquescent pneumonia.
	7.5 per cent.			
51–60 years	9	5	2 [5]	[5] *a.* Male, 54 years, very intemperate; probably complication with epidemic cerebro-spinal meningitis. *b.* Male, 55 years; delirium tremens.
	7.0 per cent.			
61–70 years	6	4	4 [6]	[6] *a.* Female, 69 years. *b.* Male, 70 years; pneumonia of doubtful importance in connection with cardiac valvular insufficiency and hemiplegia (embolism). *c.* Male, 64 years; total obliteration of both pleural sacs. *d.* Male, 69 years; terminal pneumonia in carcinoma ventriculi.
	5.0 per cent.			
71–90 years	2	5	4 [7]	[7] *a.* Female, 74 years; terminal pneumonia in chronic peritonitis. *b.* Female, 81 years. *c.* Male, with marked atheroma of arteries; temperature, 102.2° F., with pulse 30. *d.* Female, 79 years; marked atheroma and mitral insufficiency.
	4.0 per cent.			
93 years	—	1	0	
Total	114	86	24	
	200			

TÜBINGEN POLYCLINIC.

Classes by Age.	Sex.		Deaths.	Remarks.
	Male.	Female.		
Up to 1 year	1	2	0	Cases of pneumonia in the Tübingen polyclinic from May, 1873, to beginning of June, 1874.
1–5 years	5	3	0	
6–10 years	3	4	0	
11–20 years	3	1	0	
21–30 years	3	2	1[1]	[1] Female, 28 years; double croupous pneumonia and recent pleuritis; was confined a few weeks previously; large pleuritic effusion on the right side; the effusion was incompletely absorbed and, as the autopsy showed, had destroyed a part of the right lung; very marked struma.
31–40 years	1	2	0	
41–50 years	3	0	0	
51–60 years	1	4	3[2]	[2] *a.* Male, 55 years; complication with general dropsy. *b.* Female, 55 years; very weak heart, otherwise no complication; had been very badly nursed. *c.* Female, about 60 years; died from debility after the pneumonia had anatomically almost entirely disappeared.
61–70 years	4	4	2[3]	[3] *a.* Male, 63 years; double croupous pneumonia; "calcareous metastasis" of the smaller arteries of the brain. (Hasse, Nervenkrankheiten. 2. Auflage, page 567.) *b.* Female, 63 years; terminal pneumonia in carcinoma uteri.
71–80 years	1	1	0	
Total	25	23	6	

I desire to say once more, that in my remarks on the treatment of pneumonia I have not intended to lay down a plan of treatment which is to be employed under all circumstances. I

have merely tried to find the constant quantity which figures largest in the problem of the danger attending this disease. This quantity may be so small that in a given case it may be disregarded, and in the great majority of cases among children and vigorous persons this is actually the case. Such individuals recover without requiring treatment, unless antipyretic measures be adopted for prophylactic purposes, as I have suggested. Each patient must be judged by himself, and the necessity for attending to the peculiarities of the case is far greater in pneumonia than, for example, in the treatment of typhoid fever by the abstraction of heat. But for the future one point must be regarded as settled, viz., that all prostrating measures, and all medicines which tend to paralyze the heart, when given in doses sufficient to reduce the temperature, should be excluded from the treatment of pneumonia. So also any mode of treatment, which urges the heart to excessive action, is wrong, because the over-stimulation results in paralysis. That patients get well with all possible "methods," is obvious; the fact only shows that pneumonia is a typical disease, and that man is an animal very tenacious of life; nothing more and nothing less.

To conclude; in the milder cases there is no positive necessity for any treatment except a simple regulation of the diet. But the prudent physician will even here usually prefer active interference to passive expectation. In severe pneumonia very much may be accomplished by treatment, if it be carried out in strict accordance with the principle that in this infectious disease it is the heart whose activity must be maintained against the evils which directly threaten it.

The treatment of the complications and sequelæ requires mention. It is hardly to be expected that we can obtain a single point of view in this case. There is no longer a single morbific cause in action, but a variety of them. In regard to the complications, it is generally necessary to distinguish whether the pneumonia or the coexisting disease be the controlling factor. This question does not admit of a universal answer, but is to be decided for each case by itself, and even then sometimes not until the autopsy. The treatment will, of course, depend upon the determination of this point. It is evident, therefore, that it

is useless to discuss the therapeutics of complications, as it is impossible to mention every contingency which may arise, and vague suggestions are of no use to a physician.

It is different, however, in regard to the sequelæ. In cirrhosis, gangrene, and perhaps also abscess, there is an empirical remedy, oil of turpentine, whose efficacy is not to be gainsaid. I have used it upon the recommendation of Huss[1] in a great number of cases, and generally when the local symptoms have continued longer than usual after the pneumonic infection has run its course. I am unable to present numerical evidence of its therapeutic properties, but it is certain that it diminishes the quantity of secretion from the bronchial mucous membrane. I have measured the quantities discharged in chronic cirrhosis with bronchiectasis, and in all catarrhs when they had passed the acute stage, and have found the result constant. It is, of course, conceivable that the medicine was successful because it was used when the pneumonia was slowly resolving. In pulmonary gangrene the use of turpentine has, as is well known, been recommended, especially by Skoda. I do not regard the inhalations, the mode he generally used, as any more efficacious than the internal administration. I prefer to order the pure oil of turpentine in doses of twelve drops six times daily, mixed with from three to five ounces of milk. This is the form which is most agreeable to the patient, and least troublesome to the digestive organs. If the larger doses cannot be borne, we must give smaller ones, and gradually increase them. If one prefers to give the medicine in gelatine capsules, the subsequent drink of milk should not be forgotten, because by its use the simplest form of emulsion is obtained. The officinal emulsions are far less serviceable.

It is hardly necessary for me to add that the maintenance of the patient's strength by tonic treatment is all-important. This should not be carried out in a routine manner, but should be adapted to the special requirements of the case.

[1] L. c., p. 175, recommended for "gray hepatization."

CATARRHAL PNEUMONIA.

(SYNONYMS: BRONCHOPNEUMONIA; LOBULAR PNEUMONIA.)

GENERAL LITERATURE.—*Barthez* et *Rilliet*, Traité clinique et pratique des maladies des enfants. Paris, Baillière, 1853, II. édition.—*Bartels*, Virchow's Archiv, Bd. 21, 1861.—*Ziemssen*, Pleuritis und Pneumonie im Kindesalter, l. c., and *Ziemssen* und *Krabler*, Klinische Beobachtungen über die Masern und ihre Complicationen. Danzig, Ziemssen, 1863.—*Steffen*, Klinik der Kinderkrankheiten. Berlin, Hirschwald, 1865.—*Barrier*, Traité pratique des maladies de l'enfance. Paris, Chamerot, 1861, III. édition.—In addition the treatises on diseases of children, by *Gerhardt*, *Hennig*, *Steiner*, *Vogel*.

HISTORY.—*Aëtius*, "Medicæ artis principes." Paris, H. Stephanus, 1567.—*Morgagni*, "De sedibus et causis morborum." Lugd. Batav., Hank, 1767.

ANATOMY.—*Friedländer*, "Untersuchungen über Lungenentzündung." Berlin, Hirschwald, 1873.—See also under the head of Croupous Pneumonia.

HISTORY.

IT was not until late in the history of medicine that the distinction between catarrhal pneumonia and other forms of pulmonary inflammation was positively and clearly defined. Though the more intelligent physicians were aware of the difference between the pneumonia of the aged, and that which occurred during the age of vigor, we should not be justified in attempting to establish a claim of identity between the pleuritis of Aëtius, or the peripneumonia notha of Sydenham, and our catarrhal pneumonia. Cases of the latter were certainly designated by the former terms; but so also were cases of croupous pneumonia, and probably of pleuritis, according to our modern conception of them. The pneumonia of children after measles, etc., is nowhere described as an independent affection. This is, perhaps, the best evidence that the state of knowledge concerning catarrhal pneumonia was very defective.

Probably the earliest record worthy of note in this connection is found in Aëtius of Amida (about 550 A.D.). The paragraph[1] bears this heading: "De ea quæ pleuritis esse putatur, quum tamen non sit." The term "pleuritis notha" is first found in the index, and therefore is certainly older than Sydenham.

"Out of most of the 'crudities,' crude, mucous, and tenacious 'humors' are generated in the body, which sometimes discharge themselves into the cavity of the chest, or also into the lung. As they thus, by accumulation, distend the membrane which lines the thoracic wall internally, they give rise to pain which, to the inexperienced, simulates the development of an inflammation. They do, however, by invading the air-passages, likewise cause dyspnœa. . . . . It is requisite, therefore, to observe carefully the character of the respiration, to examine the pulse, and to examine by touch the kind of heat. For in such cases the respiration is not rendered difficult by pain, as is otherwise so commonly the case, but rather by obstruction of the air-passages through secretions, and respiration is performed in the same manner as if the neck were constricted by a rope. These patients almost always lie flat on their backs, because they are weak, and they are likewise unable to speak distinctly. They usually have no fever; sometimes indeed their bodies feel cold. If they are attacked by fever, it is very slight, the patients also having little thirst. The best evidence on the subject [of fever] is this: The pulse is slow, it is very small and quite sluggish; altogether the opposite of what we usually find."

In the treatment, venesection is entirely discarded; indeed its profuse employment is denounced as directly promoting a fatal issue. On the other hand, an evacuating method is recommended, cataplasms to the chest, emetics in case of threatening suffocation; for severe cough, pains, and insomnia a remedy containing narcotic substances (antidotum isotheon); and during convalescence stimulating diet and wine.

The most essential part of Sydenham's[2] description is as follows: "At the commencement of winter, often also towards its close, and even at the beginning of spring, there is an annual outbreak of fever, which has not a few of the attributes of pulmonary inflammation. This chiefly attacks adults and strong individuals, who have already attained adult age, or also, still more often, such as have passed it, and those who are too much addicted to the use of spirituous liquors, especially brandy. . . . . In the first onset of the fever the patient is alternately feverish and chilly. He is dizzy, and complains of cutting headache as soon as the cough attacks him; he vomits all liquids, either during a coughing fit or without it; his urine is turbid, and very red; the blood taken from the body resembles that which is taken when there is pain in the side. Very often also dyspnœa is added, when he respires quickly and often. When he is urged to cough, his head feels as if it would burst; for so the patients generally express themselves. The whole chest is painful, or at least bystanders hear a rattling in the lungs as often as the patients

[1] Tetrabiblion II., Sermo IV., Cap. 68, p. 434 of the edition cited.

[2] L. c., Vol. I., Sect. VI., Cap. 4. pp. 303, 304.

cough, because the lung cannot expand itself sufficiently, and because, as it seems, the air vesicles are very much compressed by the swelling, whereby the circulation of the blood is prevented, and there is, so to speak, a condition of suffocation, although almost no signs of fever are present." "I believe that the disease is almost of the same character as the pain in the side, and differs from the latter only in that the entire lung becomes inflamed." In the treatment, venesection is employed with great reserve; purgatives are substituted in its stead. Habitual brandy-drinkers must be gradually weaned from its indulgence.—Van Swieten[1] discusses pneumonia notha in a more detailed manner. From the chapter on diagnosis I quote:

1. The body of the patient must be encumbered by "lenta mucosa saburra."
2. Complaints of oppression and pressure in the chest.
3. Slight fever, initiated and accompanied by frequent rigors.
4. Behavior of the respiration, of the cough, of the urine, etc., is described exactly, in great part with literal citation, according to Sydenham. The only original observation is that the urine in isolated instances scarcely appears changed. The treatment is that recommended by Sydenham, Progress is not observable here, only a further development of what the great English physician had previously expressed.

The detailed descriptions in Morgagni[2] show most plainly how incomplete the symptomatology of pleuritis "or peripneumonia notha" appeared to the physicians who flourished before the introduction of the methods of physical examination. He endeavors to determine rules for differential diagnoses. It appears to me to be beyond doubt that the pneumonias of the aged, especially when they were associated with some bronchial catarrh, were designated as "nothæ." Among Morgagni's autopsies[3] I find one single case of catarrhal pneumonia in a female child fourteen days old. No remark is made concerning any deviation from the post-mortem appearances in other cases.

Nor has catarrhal pneumonia received its proper share of attention in later times. J. P. Frank,[4] treating on the subject of "peripneumonia notha," speaks only of a severe catarrh of the bronchial ramifications. A. G. Richter[5] expresses himself in a similar manner.

Even Laënnec[6] does not sharply define this form of pneumonia. He treats of it as "catarrhus suffocativus," or as small diffuse deposits of pneumonic infiltration which are complicated by the above-mentioned form of catarrh, and are difficult of recognition. This is evident, especially from Laënnec's case cited by Andral,[7]

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[1] L. c., Vol. II., 837-74, p. 799 of the edition cited.
[2] L. c., Epistol. XXI., 11-16. Compare also Epist. XIII., 2, 3.
[3] L. c., Epistol. XX., 15.
[4] L. c., Vol. I., pp. 131-188.
[5] L. c., Vol. I., p. 425.
[6] L. c., Vol. I., p. 157, and p. 338 et seq.
[7] Clinique médicale, Vol. I., p. 384, Observat. 46.

which was certainly a catarrhal pneumonia. Nor has Andral differentiated this form, as plainly appears from his remarks concerning the diagnosis in connection with the above case. The same applies to Stokes.[1] He settles the question with the remark that acute primary bronchitis may pass into congestion and inflammation of the lung.

It appears that French physicians who treated diseases of children were the first definitely to separate catarrhal pneumonia from the croupous variety.[2] Barthez and Rilliet[3] give a good description. Not having had sufficient access to the French literature on the subject, I am unable closely to follow its progress. Grisolle's work contains a very voluminous bibliography. He mentions Gillette and Roccas as men who correctly apprehended the significance of senile pneumonia. Among the Germans, Bartels[4] and Ziemssen[5] have recently furnished the best works. Steffen's treatise[6] is also worthy of mention.

ETIOLOGY.

Catarrhal pneumonia is always a secondary morbid process. It never originates primarily in the alveoli, being usually preceded by an inflammation of the bronchial mucous membrane. It is only when an irritant, such as chlorine gas, for instance, has exerted its action simultaneously on the alveoli as well as on the bronchi that inflammation arises in both at the same time. By prefacing our remarks on etiology with this proposition we at once limit the subject to its proper sphere.

Anything which causes catarrh may also produce catarrhal pneumonia. The more frequently any given morbific agent produces bronchial catarrh, the greater is the probability that this agent also favors the genesis of catarrhal pneumonia. These are simple but necessary deductions from the preceding proposition;

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[1] L. c., p. 82.

[2] Compare Grisolle, l. c., p. 406.

[3] L. c., Vol. I., p. 497 et seq.

[4] Remarks on an epidemic of measles observed in the Polyclinic at Kiel during the spring of 1860, with special reference to the pulmonary affections associated with it. Virchow's Arch., Vol. XXI.

[5] Pleuritis und Pneumonie im Kindesalter.

[6] Klinik der Kinderkrankheiten, Bd. I.

or, more correctly, these are the facts culled by experience which warrant the announcement of this proposition.

The etiology of catarrhal pneumonia is therefore partly allied to that of catarrhal bronchitis. We have only further to inquire into the circumstances which determine the development of a pneumonia during an already existing bronchitis.

If it were the intention to give absolute statements as to the frequency of catarrhal pneumonia, its geographical distribution, etc., a series of antecedent questions would have to be answered, which in part have not, as yet, even been formulated.

It would be necessary to know how many individuals in a community of definite size, and during a somewhat lengthened period of time, have suffered from diseases of which bronchial catarrh is a symptom; how these catarrhs are distributed over the different ages, and whether there are differences in this respect according to the primary disease; again, with what frequency catarrhal pneumonia has occurred at different periods of life, and various other matters. The material existing at present does not allow us to formulate such statistics. A general statement must therefore suffice.

It is difficult, from the communications at our disposal, to estimate definitely in figures *the frequency of catarrhal pneumonia after certain diseases which are associated with catarrh.* In the majority of the tables contained in the literature of the subject, authors have neglected to state the number of cases of the primary disease which have been observed by them during the time specified, aside from those which were complicated with catarrhal pneumonia. It is evident that this point is of decisive importance, since these primary diseases are chiefly of an epidemic character. The physician who by chance has not had occasion to observe either measles or whooping-cough during a series of years, will calculate their frequency at a much lower rate than another who has had much to do with these diseases. In the absence of the basis mentioned, here too the correct ratio can only be approximated.

Experience has proved that catarrhal pneumonia is very apt to occur after the *infectious diseases* which are combined with bronchial catarrh. Among these are measles, whooping-cough,

diphtheria, influenza, rubeola (*Rötheln*); less frequently, typhus, variola, and scarlatina. Among the acute, non-infectious disorders, genuine bronchial catarrh must be designated as the more frequent cause.

Of the *chronic diseases* which frequently predispose to this affection, are rhachitis, and then the anatomically manifold group of infantile atrophies. In the latter instance, however, it must be observed that the development of a catarrhal pneumonia is determined by the accidental accession of a bronchial catarrh. Lastly, we must refer to the occurrence of catarrhal pneumonia in connection with *tuberculosis*. Pulmonary implication in the acute miliary tuberculosis of childhood is probably always associated with catarrhal pneumonia.

It is self-evident that *every disease of an organ which either directly or indirectly favors the development of bronchial catarrh*, must also be recognized as the indirect cause of catarrhal pneumonia; thus, emphysema, cardiac disease, etc. *Traumatic influences, in so far as they determine bronchial inflammation*, are likewise to be included in the category of exciting causes: such are *foreign bodies in the bronchi*, and the *inhalation of gases* which excite inflammation (chlorine, ammonia, etc.).

In the above an approximative calculation of the frequency of causes has been attempted and arranged in that order. A somewhat more exact statement, in the form of figures, might, perhaps, be compiled from the reports of epidemics. I must confess, however, that the positive result which might thus be obtained, would hardly be proportionate to the labor required. For the purposes of this work a mere estimate may suffice.

Age is an *individual* factor of great importance. The extremes—the period of childhood and that of old age—are peculiarly disposed to catarrhal pneumonia. The age of vigor, in the main, remains exempt.

In order to substantiate the general truth of this assertion, we have only to appeal to the unanimous verdict of all experienced physicians. Only under very extraordinary circumstances does catarrhal pneumonia occur during the full bloom of mature life. It is much more difficult to ascertain the precise distribution of this disease over the different ages; for the dis-

position of the several ages to the primary diseases is very variable. Experience teaches that children under six months of age are rarely attacked with measles. But in an individual of such tender age, an intense bronchial catarrh will certainly call forth a pneumonia more easily than in one of the age of ten years, provided the patient does not succumb before true catarrhal pneumonia could develop, the vitality and power of resistance being already overpowered by the catarrh. Hence, the factor which varies for every primary disease, namely, the disposition of the several ages to this disease, engenders exceedingly complicated relations. These can, of course, be unravelled and arranged in the form of statistics, provided the material is sufficiently abundant. In this case, however, we can make an approximative estimate without entering into a mass of details. *All observations agree, that the first three years of life are those which are most often attacked by catarrhal pneumonia.*

Among 98 cases of catarrhal pneumonia, Ziemssen reports 67 as having occurred before the third year; Steffen 52 in 72 cases during the same period. Bartels states that in 68 cases of pneumonia during measles, 42 occurred before the fifth year.

So far as we can judge from the existing data, *sex* has no influence on the frequency of catarrhal pneumonia.

The influence of *constitution* cannot be established with certainty. It may be said, in general, that a strong constitution offers a certain degree of protection.

The term, good or bad constitution, when applied to childhood, of course includes, just as in later years, the primary predisposition as one of the chief factors. Aside from this, however, *external influences*, such as nourishment, the air which the child breathes, the care bestowed on it, exert a peculiarly strong influence at this early period; and in catarrhal pneumonia especially these influences are unusually active.

Children in overcrowded apartments, who are forced to breathe air pregnant with all kinds of miasms, are much oftener attacked than those who live under more favorable conditions. In this respect I believe that all experienced physicians will agree with Bartels,[1] who lays great stress on these points. Dur-

[1] L. c., p. 136.

ing every epidemic of measles or pertussis there are more cases of catarrhal pneumonia in public than in private practice. For the same reason pulmonary affections are more numerous and severe during epidemics of these diseases in winter than in summer. For during the winter ventilation is neglected.

Derangements in the bony walls of the thorax or in the respiratory muscles, which prevent the complete expansion of the lungs, are of great influence on the genesis of catarrhal pneumonia.

Every unbiased observer will admit that the accession of a *cold* to an already existing catarrh does not favor the development of a catarrhal pneumonia. It is different when we look upon the *cold* as the exciting cause of the catarrh which must necessarily precede the pneumonia. Individuals whose *locus minoris resistantiæ* is located in their bronchial mucous membrane, may become affected with a catarrh after taking cold, to which, under otherwise favorable conditions, a catarrhal pneumonia may accede.

But it cannot be maintained that the influence of a cold extends beyond this,—at least not for the acute catarrhs which are combined with fever. A patient with fever does not take cold.

PATHOLOGY.

Symptomatology, Course of the Disease in General.

I agree with Ziemssen in distinguishing two forms: one, acute, and the other characterized by a slower course. As typical illustrations of these we may take the catarrhs of measles as exciting causes of the first, and bronchitis with pertussis as the starting-point of the second. The symptomatology within each of these two groups is very similar and very uniform. The two classes, however, differ very much from one another; not essentially, but in their forms of manifestation.

Acute catarrhal pneumonia originates in a very intense and widely diffused catarrh of the smaller bronchi. In the beginning the picture differs but slightly from that presented by bronchitis, and the most striking features of the latter remain promi-

nent. Rapid, superficial respiration with playing of the nostrils, and slight moaning, the face reddened and cyanotic, the eyes glistening, and frequent cough accompanied by expressions of pain. The thoracic movements are also identical with those of capillary bronchitis; with every inspiration the lower ribs are markedly retracted; they are strongly drawn inwards whenever, previous to coughing and crying, the patient takes a deep inspiration. But as soon as the pneumonia has assumed greater proportions, the scene changes.

Then,—and this is one of the first symptoms,—the cough ceases, or at any rate becomes less, and is also less liable to occur in prolonged paroxysms. It seems to be much more painful, for every effort at coughing is accompanied by moaning or crying, and this, perhaps, is partly the reason why the paroxysms are less prominently marked; an effort of the will prevents them. Soon great restlessness is added. No position is maintained long; the patients toss about, are capricious in their demands, dissatisfied with everything, and do not allow their attendants a moment's rest. The respiration continues to grow more superficial, the redness of the face diminishes and gives place to a more distinct blue color, which then also fades into a dead white, the cheeks alone contrasting strangely by a few intense bluish-red spots. The mucous membrane of the lips becomes very cyanotic; and if the epidermis is delicate, thickly swollen, bluish-black venous cords are seen through the skin. The picture of carbonic-acid intoxication now develops. The noisy restlessness diminishes, the patients lie in an apathetic condition, from which they are aroused for a short time only by the paroxysms of cough, which continue to grow less frequent. But the body is not actually at rest for a moment. There is tossing about, frequent scratching of the skin, even perhaps to actual laceration, low moaning and sighing. Coarse and fine mucous râles are heard with every respiratory movement. Frequently, at the close of the scene, there are cerebral symptoms, not only a deepening of the apathy into coma, but the whole train of symptoms which usually accompany a tubercular meningitis. *Death* may occur very suddenly with convulsions, or very slowly. A *change for the better* always takes place gradually, never in a short

time. Transient or permanent relapses which re-establish the entire disease, are not infrequent. Aside from these relapses, the course of the disease, whether its result be favorable or unfavorable, is a comparatively rapid one; within from one and a half to two weeks the danger is either removed or death has resulted. The *local restitution* requires a much longer time; it is very commonly associated with more or less marked symptoms of general derangement, especially anomalies of temperature.

As a rule there is high *fever* in this form of catarrhal pneumonia, the temperature being about 104°, or even 105.8° F., without any characteristic type. It never falls to its normal standard in a few days or hours. In most cases the local pulmonary changes develop rapidly; examinations from day to day usually reveal the progress of consolidation.

The *form of catarrhal pneumonia* which is *characterized by a slower course* differs essentially from the first in its very gradual development, with moderate febrile action, during which the symptoms of carbonic acid intoxication become more prominent. Children suffering from whooping-cough, or a general diffuse catarrh running a subacute course, gradually cease coughing as the pneumonia develops. They become peevish, apathetic, slumber much, have no appetite, indeed often very decidedly refuse all offers of nourishment. The attentive observer is struck with the tired expression of countenance, the superficial respiration, the pale complexion, with its marked blue shading. As a rule, however, no special change is observed by the bystanders; they may perhaps be pleased that the cough has diminished. In most cases the physician is first called when the excessive emaciation awakens the anxiety of the most careless observer, or when a stronger febrile exacerbation simulates the invasion of a new affection. The further course of the disease is characterized either by a very gradual death with distinct symptoms, during the last days, of the excessive accumulation of carbonic acid, or again, an exacerbation calls forth the picture of an acute catarrhal pneumonia. Recovery rarely takes place without permanent damage to the lungs; some traces probably always remain. In this form the duration of the disease must be measured by months instead of by weeks. It is subject to all the changes

which are liable to befall a system suffering from a severe attack of disease in a vital organ.

In this form of catarrhal pneumonia the *fever* is milder, very changeable, and devoid of any regularity. Towards the end considerable elevations of temperature may set in. The *local changes* develop very gradually, but they usually attack a much larger area of the lung than in acute pneumonia.

Having reviewed the appearances presented by catarrhal pneumonia in childhood, it still remains to add a few words concerning its occurrence in adults. I consider it unnecessary here to enter into details. On the whole, the processes above described are repeated, and having studied the disease in childhood, we have seen it presented in its most varied aspect as well as in its most practical light. Every physician who understands catarrhal pneumonia as it occurs in childhood, will be able to appreciate the changes in the symptomatology which are determined by age.

In strong individuals in the vigor of life I have most often seen catarrhal pneumonia in connection with *diphtheria*. These forms, which, as a rule, run a very acute course, present the additional symptoms peculiar to intoxication from carbonic acid. But, since very diffuse diphtheritic bronchitis may produce similar effects, it is necessary to resort to physical examination as the only certain means of detecting the pneumonia.

My experience leads me to believe that this will generally be found to be true in *adults, provided their health has not been much deteriorated* from any cause. For, in them, a very intense and diffuse bronchitis is requisite to call forth catarrhal pneumonia. But a bronchitis of this character is always supplemented by symptoms of the most marked carbonic acid intoxication.

The aspect of the case differs when it concerns an *organism debilitated in any manner*, whether the diminished power of resistance be due to antecedent acute febrile disease or a chronic error of nutrition. Such cases are infrequent. The few which I have observed were not entirely pure cases, but were complicated with hypostasis. In these the deleterious action of the carbonic acid was rather subordinate. Physical examination of the lungs alone determined the diagnosis.

Many of the so-called "passive infiltrations" which are the cause of death among the *aged and cachectic*, should be classed under the head of catarrhal pneumonia. We know how obscure their symptomatology is. It is likewise very difficult to recognize, by any variation in the picture presented, those instances of catarrhal pneumonia in the aged which are associated with very diffuse bronchial catarrh. Furthermore, physical examination more often fails in these cases. For it is uncommon to find large accumulations, and the small, very diffuse masses which are distributed throughout both lungs simultaneously, though they may be numerous, do not determine demonstrable changes in resonance. Still less can be expected from auscultation, on account of the rhonchi. The *traumatic forms* which originate from the inhalation of irritating gases or from the intrusion of foreign bodies into the bronchi, probably develop more rapidly than any other form of catarrhal pneumonia.

Anatomical investigation teaches that these pneumonias—though superficially most closely resembling the croupous form—are not croupous in character.

Pneumonia produced by inhalations is more similar to very diffuse, acute, capillary bronchitis, and in such cases, where the poisoning of the system is very intense, the disease becomes lobular immediately. In the pneumonias from *intrusion of foreign bodies*, on the other hand, infiltration of an entire pulmonary lobe is sometimes found, and this may develop very rapidly.[1] In other cases—the nature of the body introduced into the bronchus probably determining the difference—a more difuse extension of the pulmonary affection results. The course and symptomatology of the disease are subject to much variation.

Anatomical Changes.

The macroscopic appearances will first engage our attention. In subjects who have died from catarrhal pneumonia, a strongly developed bronchial catarrh is always found. As a rule, this catarrh is encountered as high up as the trachea and larynx,

[1] Compare *E. Wagner*, Zur Casuistik der fremden Körper in den Luftwegen, Archiv d. Heilkunde, V., 1864, p. 347 et seq.

increasing in intensity below that point as it descends, and attaining its maximum development in the lower and posterior portions of the lung. The mucous membrane is more or less intensely reddened, thickened, and disposed in folds, and sometimes pervaded by extravasations. When the catarrh is recent, as Bartels remarks, a vitreous, tenacious mucus, mixed with bubbles of air, is found on the mucous membrane of the larger bronchi; the finer ramifications, on the other hand, are free from secretion. This condition, which I have also repeatedly observed, is not common. In most cases the catarrh leads to the formation of a creamy, whitish-yellow pus, from which all air has disappeared. This then fills the bronchi down to their final ramifications. At first only the bronchial mucous membrane is attacked by the inflammation; soon, however, the latter extends to the whole substance of the bronchial tube, which then projects above the level of any section of lung tissue that may be made. When the process has been of any considerable duration, the smaller bronchi are dilated cylindrically, and are frequently filled with a yellow, inspissated secretion. The alveoli—more especially after whooping-cough—may also be dilated in globular form from the forced intrusion and caseation of the secretion (Fauvel, Ziemssen). Before arriving at the condition of true inflammation, the parenchyma of the lung passes through several stages. At first the lungs will be found to have diminished in volume at their margins, and to have become wrinkled and flabby, so that the edges can be turned in, and this condition, as a rule, exists symmetrically on both sides. The color —which either shades on a blue tint or is downright intensely blue—presents such a striking contrast to the surrounding tissue, that this is even more remarkable than the change in the level of the section. When these portions are incised, dark blood in sparse amount exudes. All air has been expelled from them. Ecchymoses under the pleura, from the size of a pin's head to that of a nut, and in variable number, are almost constant. The portions thus affected can be easily reinflated by blowing a current of air through the bronchi. The condition described may properly be designated as *simple collapse of the lung*.

This collapse may attain a very considerable extent. The

entire lower lobe, very commonly also the tongue-like expansion of the left upper lobe, may be, and not infrequently are, affected. *The process always progresses from behind and below forwards and upwards.*

In the more sluggish forms an entire lung may gradually pass into a condition of collapse.

Together with the pulmonary collapse, we invariably find emphysema more or less markedly developed according to the extent of the collapse. When both lower lobes are atelectatic, then both upper lobes are usually inflated to such a degree that, by overlapping, they almost entirely conceal the pericardium. They almost touch each other, and do not diminish in size after the thorax has been opened. When the collapse is less extensive, the emphysema is generally confined to a smaller portion of the lung in the neighborhood of the collapse.

If the process advances to inflammation, we then find, first, isolated nodules, from the size of a pea to that of a hazel-nut, within the collapsed portions, which can easily be distinguished by the touch, and are slightly elevated above the surrounding surface. Adjacent infiltrations gradually become confluent, and in this manner the process may extend over an entire pulmonary lobe. Small isolated portions which are still accessible to air, and can be rendered more apparent by inflation, may almost always be found within the inflamed parenchyma. But the latter itself cannot be inflated, because all the air has been expelled from it. On making a section of the tissue, it first presents a brownish-red color, which, after temporarily assuming a more distinctly dark-brown hue, eventually passes into a grayish-red. *Granulation is never observed;* on the contrary, the section presents a smooth and uniform appearance throughout.

If the bronchi are filled with pus of a thicker consistency than usual, the pus which escapes from the finer bronchi in spherical masses may impart an appearance to the surface which, at the first glance, resembles tubercles. The tissue which is the seat of pneumonic infiltration is soft and friable, and the fluid contained in it is tenacious and turbid. The discoloration which exists always begins in the centre, and spreads towards the periphery of the lobules. In case the process ad-

vances still further, the pulmonary tissue appears of a pale blue color, very tough, hard, and homogeneous. The bronchi are filled with inspissated exudation, the connective-tissue structure of the lung is increased, so that a section presents thick, whitish-gray, intersecting bands of fibres. I dissected a case of this character eight years subsequent to the primary disease (measles), and I found complete contraction of the lung, with the development of bronchiectasiæ, strong pigmentation of the newly-formed connective tissue, and consecutive hypertrophy and dilatation of the right ventricle. At other times degeneration of one or more cheesy infiltrations results; the lung-tissue immediately surrounding them is then destroyed, and cavities are formed.

In the beginning the *pleuræ* are found only slightly injected, the dark red spots of ecchymosis standing forth in marked contrast to the general surface. Soon, however, this is covered with a thin layer of fibrine. If the inflammation of the lung persists, this layer continues to thicken, and the costal pleura likewise participates to a greater extent in the pathological changes. Finally, adhesions result, which are always more or less disposed in layers, and cause the formation of a thick callosity. Free effusions are certainly not frequent.

The *bronchial glands*, even in recent cases, are swollen, juicy, and hyperæmic.

To recapitulate: *The diffusion of the process over the lungs in the form of isolated infiltrations, and the coexistence of the most different stages are characteristic phenomena. The lung, when the seat of pneumonic-catarrhal disease, first diminishes in volume, then exceeds its normal size, and lastly returns to it. Individual portions, and even entire lobes, may remain, for months at least, in an atalectatic condition. Inflation is the best means for distinguishing truly inflamed from simply collapsed portions.*

Acute miliary tuberculosis must be mentioned as a not infrequent complication. Post-mortem changes in other organs are not constant.

In my description I have essentially followed that of Bartels, whose statements I have verified by numerous autopsies. I have likewise frequently availed myself

of Ziemssen's description. The views of these two authors correspond completely, and are triumphantly vindicated by those observations on the dead body which every man is obliged to undertake who aspires to rank as a competent judge of this question. I write under the recent impression of an extensive epidemic of measles, which is still in progress; nothing essential, however, in my opinion, could be altered in or added to the above descriptions.

The *more delicate changes* which take place in catarrhal pneumonia have recently been studied by numerous and excellent observers. We are fortunately enabled to observe these processes from their very initial step, as the morbid condition we are considering can easily be excited for purposes of experiment.

That form of pneumonia which occurs after section of both pneumogastric nerves (Traube), being engendered by the intrusion of the oral fluids into the bronchi, is a catarrhal pneumonia of an exquisite character. Its behaviour corresponds in every detail with acute catarrhal pneumonia, as we observe it in children after diphtheria and measles. Friedländer, whose description seems based on exceedingly accurate investigations, distinguishes it as follows:

At first hyperæmia of, and sero-sanguineous exudation into, the alveoli take place; then, while the epithelium of the alveoli swells, numerous lymphoid cells appear in the blood-vessels, in the interstitial connective tissue, and in the alveoli. The lymphoid cells come from within the blood-vessels; there is an emigration, as will be found in every acute inflammation. The alveolar epithelial cells take no *active* part in the process; at any rate no such action is demonstrable. They undergo merely a passive change, take on swelling in the transuded serum, and at a later period partially succumb to fatty degeneration.

Buhl's[1] conception differs in some essential particulars. He concedes that the contents of the alveoli, so far as they consist of pus-corpuscles, may have originated on the spot, and indeed by emigration from the capillaries. He questions whether the mucus which is found in the alveoli has the same origin; for, admitting the further possibility that the alveolar epithelium might produce mucus, such has not been proved. In Buhl's

[1] Zwölf Briefe u. s. w. 2. Brief.

opinion it is an established fact that by far the major portion of the catarrhal product contained in the alveoli has been carried there by aspiration. He admits that an inflammatory irritation may likewise be set up in the pulmonary tissue at a later period, in case this state of things is long continued.

There does not seem to me to be any irreconcilable antagonism between these two views. I hold that Friedländer's experimental investigations have proved, that in the form which he has studied pneumonia after section of the pneumogastric, emigration *in loco*, *i.e.*, into the alveoli, does in fact take place. As in other respects the lesions are in entire conformity with those found by Colberg[1] in the recent catarrhal pneumonia of children, I cannot conceive why the emigration of the white blood-corpuscles should not take place in man as well. For this is a process that may invariably be observed wherever inflammation occurs. If in this case emigration did not take place, the obstacle which prevents the operation of a universally valid rule would have to be demonstrated. As long as this is not done, no one will be convinced by a mere denial. On the other hand, however, it is extremely probable that during the excessive respiratory movements (cough), which so regularly attend the development of a catarrhal pneumonia, the bronchial contents enter the alveoli and finer bronchi from the superior portions of the respiratory passages.

Whether the epithelium of the alveoli—even in cases which run a slower course—participates only in a passive manner, has indeed not yet been positively determined. Good authorities (Rindfleisch)[2] attribute to it an active interference, proliferation, etc.

Regression to the normal condition takes place by the formation, firstly, in consequence of fatty degeneration, of a reabsorbable emulsion, which is then really in part absorbed and in part expectorated. The anatomical description of the further development of the process into cirrhosis or phthisis is not included in the task here allotted to me. I must therefore refer to the

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[1] Deutsch. Archiv f. klinische Medicin, Bd. II., 1866, p. 453.

[2] L. c., p. 355.

appropriate sections touching on these points in other portions of this Cyclopædia.

Analysis of the Symptoms.

In considering the febrile relations of the disease under consideration, it should first of all be premised that *catarrhal pneumonia has no regular type of fever*. Experience has established this fact, which is in full accordance with the views I have already expressed under the head of croupous pneumonia, concerning the significance of febrile types. Here again we have two factors which influence the course of the temperature, viz.: First, the tendency of normal regulation to retain the temperature at about 99° F., and so to distribute it over the period of twenty-four hours that the higher grades will occur during the day and the lower at night; and second, the factor generated by the pathological condition present. Here the condition is one of local inflammation, and not a general affection. This topical disease attains its completion in a gradual manner; it advances piecemeal. The influence exerted by it on the temperature of the body must accommodate itself to its behavior otherwise, and likewise be of variable intensity. *Ceteris paribus*, the derangement of temperature occasioned by the local disorder must be proportionate to the extent of this disorder and the rapidity with which it spreads. Let us consider the teachings of observation. The acute form of pneumonia, which runs its course rapidly, likewise has the highest range of temperature. In the subacute form the thermometer shows lower grades. But the diurnal variation is regular in neither, the maximum sometimes occurring in the morning, sometimes at night; in short, the variations are so great that in this respect one case bears no resemblance to another. It would therefore be a fruitless task to attempt the establishment of a scheme for the thermometric deviations.

The *absolute heights* reached may be very considerable. In pneumonia, during measles, a temperature of 105.5° F. is not uncommon. This may continue for days, even for one or two weeks, with but slight remissions. It is otherwise in catarrhal

pneumonia which runs a slow course. In these cases the temperature rarely attains a height of 104°—usually it is found between 102.5° and 104°—the remissions descending to 100.5°. They seldom descend lower; if they do, however, subnormal rather than normal temperatures are more frequently met with. Should phthisis develop, the temperature will naturally assume the character which is peculiar to that disease. It may be remarked, in passing, that a complete hectic-fever type is not common in children.

During the period immediately preceding death, the temperature may rise to a very considerable height, in both forms of catarrhal pneumonia. In my experience this has frequently been true. I have seen the temperature at and over 107.8° [1] shortly before death. It does happen that children die in collapse, which is associated with a low temperature of the trunk.

The catarrhal pneumonia of *old people* is characterized by only slight abnormal increase of temperature.

In *adults* the temperature may, at least for a time, reach a very high figure. But in these cases it is very difficult to determine how far the pneumonia, as such, causes the rise of temperature, inasmuch as any considerable intensity of the fundamental disease (especially if this disease be associated with fever, as in the case of diphtheria, for instance) is even more likely than in children to be responsible for the heat of the body.

The *state of the circulation* is likewise subject to considerable fluctuations during catarrhal pneumonia. It may be stated, however, that the pulse, as a rule, usually remains high, even when there is a remission in the temperature. This is an indication that the temperature here is not the only factor which determines the frequency of the pulse. But we can advance no further towards an explanation of these phenomena, for the manifold other abnormal changes—as, for instance, those of the mechanical alterations in the lungs, the interchange of gases, etc.—forbid further investigation.

In smaller children it is not very uncommon to find the pulse

[1] Compare also *Ziemssen*, p. 314; case Meyer.

too rapid to be counted; this may at least be true of the peripheral arteries even when auscultation of the heart still enables us to record the number of its pulsations. I have repeatedly counted over 200 pulsations to the minute, even when the case has had a favorable termination. In children, say up to the fifth year, the frequency will, in most cases, be found to be about 150 pulsations—the great variations in cardiac activity, which are normal at this period of life, being also perceptible under these circumstances. During these years the significance of acceleration of the pulse is of very subordinate value in determining the general condition. After the fifth year there is an essential change of the relations in those individuals only who have attained a much more advanced age. The increased irritability of the heart, however, disappears very gradually. The damage wrought in the heart by the preceding acute primary disease constitutes the essential cause of this irritability, which is the more prominent in early life, because, during childhood, the normal pulse being more rapid, the starting point of the accelerated pulse is numerically higher. This increase cannot pass beyond a certain limit. Hence the influence of a doubtful cause, or one which is not constant, will be more likely to make itself felt in the long run. Thus the man who climbs a hill but once a week may, for the first quarter of an hour, have as good wind as the native mountaineer. No rules can be laid down which are of universal application, each case being influenced by its individual circumstances. The frequency of the pulse in *old people* is not markedly increased by an attack of catarrhal pneumonia.

The *quality* of the pulse may change very considerably. In the beginning it is generally full, tense, and hard, rather than soft. But in a pneumonia of somewhat extensive development the artery becomes empty, loses its tension, and can be compressed very easily. *When the pulse becomes small and compressible it is a symptom of the most grave significance.*

Changes in the *heart*, more especially in its muscular structure, are not infrequent. They are confined to the ordinary limits: fatty degeneration occurring in affections of longer duration, and a peculiar parenchymatous degeneration in highly

febrile conditions. Probably they are never recognizable during life. We should be very guarded in drawing conclusions as to the existence of pathological conditions in the heart from derivations in the results of percussion and auscultation, because the volumetric changes in the lungs—collapse and expansion—give rise to extraordinary changes in the conducting media. The incomplete discharge of the venous trunks into the right auricle is revealed in the thickly swollen, bluish-black cords which traverse the neck, face, and chest, and do not disappear even when the patient takes a deeper inspiration.

The *relations of tissue metamorphosis* are not precisely understood. We must be satisfied with the fact that catarrhal pneumonia debilitates children to a remarkable degree. Recovery always takes place very slowly. In cases whose progress is slower, this unfavorable result is brought about by a whole series of other conditions besides the pneumonia, which are dependent on the primary disease. The appetite is diminished, often entirely suspended. In exceptional instances it happens that the children, even with high fever, continue to have a good appetite. Smaller children will take the breast or the bottle more readily than larger ones. It seems that the patients often refuse nourishment because the act of swallowing provokes severe and painful cough. Larger children express themselves directly to this effect, and in smaller children it is easily enough perceived. Vomiting not unfrequently accompanies or follows the cough. It is probably always caused by reflex irritation. And these paroxysms are sometimes followed by retching and long-continued nausea.

Diarrhœas are very frequent in catarrhal pneumonia, irrespective of the character of the primary disease from which it has developed. In the slower forms severe and prolonged diarrhœa may aggravate the danger considerably through thickening of the blood. The frequency of diarrhœa in catarrhal pneumonia might be explained on the ground of a certain degree of stasis in the intestinal mucous membrane, though at the autopsy we rarely find anything besides a very insignificant catarrh and perhaps a slightly swollen condition of the patches and follicles.

The *spleen* and *liver* present no symptoms except such as

must be considered purely accidental complications. Due allowance being made for the changes in the blood-supply, there is nothing in their condition that does not coincide with the negative results found on post-mortem examination.

In studying the symptomatology of this affection the condition of the *respiratory organs* naturally ranks first in importance, they being the organs in which those local disturbances arise which constitute the essence of the disease.

Collapse of the lung plays a very prominent rôle in the early history of catarrhal pneumonia. How is this collapse brought about? I believe the best starting-point in the discussion will be found in the fact, which is strongly and rightly urged by Bartels, that in the beginning of collapse the lower and posterior portions of the lungs are affected almost symmetrically. These are the portions which are normally subjected to the least motion. Now, when an extensively diffused catarrh has caused, firstly, contraction of the bronchial calibres by swelling of the mucous membrane, while the superficial inspirations of the febrile patient do not accomplish a complete expansion of the thorax, the vital force furnished these sections by the inspiratory muscles may become so slight that it is incapable of overcoming the elastic resistance of the lungs. If this is the case, the lung contracts to the volume which corresponds to its elastic tension. Let us assume that during inspiration muscular pressure and elasticity were equally balanced; then, when the next expiration takes place, the elasticity will act directly as the motive power, and expel a portion of the air contained in a given pulmonary segment. The air separates the opposed walls of the bronchi. These are covered by mucous membrane, which is swollen and disposed in folds. If this air be removed to the extent of allowing the bronchial walls to touch each other, the resistance against the readmission of air will increase to a very considerable degree. Indeed, it can hardly be otherwise; these projecting folds of mucous membrane are almost sure to fit into one another so closely as to produce a complete closure of the tubes, and one which is difficult to overcome. This approximation is further increased by the action of an adhesive, glutinous mucus which is secreted from the inflamed surface, and which acts just

like glue. When, therefore, the bronchial walls are once in contact, the entrance of air into the occluded portion can be enforced only by a considerable augmentation of the inspiratory pressure. Some air still remains in the occluded portion. But it is found that this also disappears when the collapse is of longer duration. This may be accomplished in one of two ways: either a portion of the imprisoned air is forced upwards, beyond the obstruction in the bronchus, by violent expiratory efforts—as by cough—or the air may have passed over into the blood. Diffusion by pressure would be necessary to accomplish this result, while simple endosmosis would scarcely suffice. A simple endosmotic equalization of gases, it is true, would deprive the confined air of oxygen and nitrogen, while it increased its proportion of carbonic acid by exosmosis. But there is no difficulty in understanding how the requisite degree of pressure might be brought about. Aside from the expiratory efforts during cough, the swelling of the alveolar epithelium, the commencing transudation, perhaps also—in case inflammation follows—the emigration of white blood-corpuscles, and the inflammatory swelling of the septa will furnish sufficient vital force for this purpose.

The description given above refers particularly to the symmetrical collapse of larger portions of lung tissue. The isolated patches of the same nature found scattered over the lungs present similar although more complicated conditions. Here, too, the process begins by the filling up or the agglutination of one or more bronchial tubes. But inasmuch as these patches are surrounded by portions of lung-tissue, which still admit of the entrance of air, it remains true that whereas, in the act of inspiration, pressure from above downwards is prevented by the closure of the bronchus, lateral pressure is not interfered with.

Let us imagine three bronchi placed adjacent to each other, with their respective alveoli, *a*, *b*, *c*, of which *b* is occluded, while *a* and *c* are free; then with every inspiration the pressure will diminish in *a* and *c*. As *b* is not accessible to air, the adjacent spaces, *a* and *c*, will experience a certain pressure by *b*, or, in other words, they will exert traction on *b*. The same effect is produced by the stretching experienced by *b* during inspiration,

as part of a structure—the lung—which is, as a whole, elastic and distended. If the vital force which is thus set free for *b* can be energized—*i.e.*, if the obstacles are not too great—the result of this action can be conceived of as showing itself only in the form of aspiration, or a suction exerted on the blood-vessels ramifying in the occluded portion of the bronchus, or on the secretion contained in its upper portion. This is true only provided the occlusion above remains intact. For it can easily be conceived that in such isolated infiltrations a giving way of the adhesions within the tube may take place by a diminution of the inspiratory pressure in the neighborhood. Precisely the same factors which are engaged in cases of general collapse likewise come into play in this instance; for in the former, too, the force of aspiration must, as a matter of necessity, make itself felt, inasmuch as we cannot imagine a complete and simultaneous closure of all the bronchial tubes. But this force is less actively engaged in the former than in the latter instances.

In collapse occurring in isolated patches the extension of the process will depend on the intensity of the opposing forces; that is to say, on the degree of vital force furnished by the inspiratory muscles and the obstacles interposed to such force, which, so far as they deviate from the normal standard, depend essentially on the catarrh. It will depend entirely on the behavior of these two factors in the different portions of the lungs, whether one or more bronchi again become pervious, or whether they are to furnish the nucleus for a further extension of the collapse. The previous collapse of a considerable portion of the lung will naturally favor the spread of this process very much. For every bronchus which in turn becomes occluded, being surrounded by respiring pulmonary tissue, is exposed to an external lateral suction power. This disappears when the portions surrounding it are collapsed, hence the obstruction of the bronchial tube need not be so firm in order to result in collapse.

Is it essential that collapse of the lung should always precede the development of a catarrhal pneumonia? If it is intended to lay down a universal rule, the question must decidedly be answered in the negative. For, as I have satisfied myself by experiment, the inhalation of chlorine gas, or rather its intrusion

into the lung, produces within a few minutes the most acute inflammation, involving the alveoli, and, as has been shown by microscopical examination (Schüppel), constituting a catarrhal pneumonia.

I am very much inclined, in explanation of the above facts, as well as of those presented by pneumonia resulting from section of the pneumogastric, to adopt the theory that *collapse results in pneumonia by reason of the presence within the alveoli of a substance capable of exciting inflammation.* The secretion of the inflamed mucous membrane may exert this effect,[1] probably with greater intensity the longer it has been exposed to the air. It is easy to suppose, then, that in those collapsed portions of lung tissue which are accessible to the catarrhal secretion, pneumonia should be developed, that is to say, that the alveolar portion of the lung should participate in the inflammation. Much can be advanced in support of this view. In the first place, I call attention to the fact that a collapsed portion of lung may remain in this condition for a long time without passing into that of pneumonic infiltration. Bartels[2] emphasizes this fact, which is by no means rare in the slow forms of pneumonia. Then again, the condition existing in cases of congenital atelectasis must be remembered. It is, furthermore, easy to understand how isolated spots of pneumonic infiltration occur in an entirely collapsed lobe; likewise, why small, completely isolated infiltrations, in the midst of tissue which contains air throughout, are a comparatively frequent lesion, even in the superior lobes. It is precisely under these circumstances that, subjected to the influence of a powerful respiratory effort, a collapsed portion of the lung, which is entirely surrounded by a parenchyma capable of inflation, is most likely to have some of the bronchial secretions sucked into it.

It is to be understood in this discussion that we do not assume it to be an absolute necessity that collapse should precede the development of pneumonic infiltration in a catarrhal affection of the lung; for the only indispensable prerequisite is

[1] This accords with Friedländer's conception, l. c., p. 30.

[2] L. c., pp. 80 and 82.

the presence of something capable of exciting inflammation in the alveoli.

The view here developed differs in various respects from those of authors generally. Ziemssen adheres more to general features, without entering much into detail. Bartels thinks that a spasmodic contraction of the bronchial muscular tissue plays a prominent part in the development of pulmonary collapse. He bases this view mainly on the fact that in recent cases very little secretion is contained in the bronchi. Hence he believes that the former theory, which accounted for the occurrence of collapse only on the supposition that the bronchial tubes were occluded by secretions, has been found by anatomical investigations to be one-sided. The fact which Bartels announces is indisputable; but I see no need of resorting for its explanation to the purely hypothetical action of the muscular structures of the bronchi; for collapse may take place in the lower and posterior portions of the lungs, as the result of defective action of the respiratory muscles alone without catarrh or any pathological change whatever.[1] The new element presented in case of catarrh, the one which presents that ready relief possible in collapse from those other causes, is the swelling and the folding of the bronchial mucous membrane. Another difficulty in accepting Bartel's theory lies in the fact that in adults, even when a severe catarrh is present, no considerable degree of collapse is liable to result.

Emphysema, or, more correctly, the acute over-distention of the lungs, which may eventually result in emphysema, is a condition which certainly originates in part during the expiratory efforts (Ziemssen). Especially will this be the case in the upper lobes. It is likewise evident that, after the collapse of certain alveoli, those adjacent to them will, during inspiration, be distended to a degree corresponding to the additional space to be filled.

Dilatation of the bronchi is likewise attributable partly to the effect of inspiration, partly to that of expiration.

At the beginning of the disease the results of *physical examination* are by no means unequivocal. On simple *inspection* it will be found that, even in case of extensive capillary bronchitis only, the ribs are drawn inward on inspiration. The more elastic the thorax, the more marked will be this phenomenon. The occurrence of collapse interferes with the entrance of air, even more than mere narrowing of the calibre of the bronchial tubes. A more marked retraction of the ribs is therefore to be looked for as soon as the lung-tissue has collapsed. On

[1] Compare the facts relative to this point under Croupous Pneumonia.

the other hand, however, it must be remembered that the inspirations will, at the same time, grow more superficial; hence, that the fluctuations in pressure within the cavity of the chest will be more circumscribed. Consequently this symptom, which is so marked, can be regarded as giving evidence only of some obstruction to the entrance of air into the lower portions of the lung. It is not pathognomonic of any special condition.

When a general expansion of the thorax takes place during inspiration, the partial vacuum thus formed within the chest must be filled. This is ordinarily accomplished by the free entrance of air. If any unusual obstacle is interposed, it may disturb the proper relation between rarefaction of the air and the equalization of pressure. Owing to the rapidity with which muscular contraction takes place at the beginning of respiration, so great a comparative vacuum is suddenly formed that the atmospheric pressure on the under surface of the diaphragm offers a degree of resistance that cannot be overcome. If the contraction of the diaphragm persists,—and, indeed, it does last longer than that of the other inspiratory muscles,—then the diaphragm changes its fixed points; the centrum tendineum, instead of being movable, becomes the fixed point, and the ends of the false ribs, perhaps even the lower portion of the sternum, are drawn towards it. The atmospheric pressure on the external surface of the thorax operates in concert with the action of the diaphragm. The analysis just given accounts, in a logical manner, for the declarations by which it was preceded.

Some conclusions may perhaps be arrived at from *palpation*. These, however, will be diametrically opposite to one another at different stages of the disease. When the lung is collapsed the heart lies in contact with a considerable extent of the thoracic wall, so that its movements may be felt through three or four intercostal spaces. If puffiness of the lungs ensues, the movements of the heart are not usually altogether lost, but they grow much less distinct, more diffused over the entire left side of the chest. As might be supposed in cases of severe catarrh, *pectoral fremitus* is very variable. The same rules which are given above, under the head of croupous pneumonia, hold good here.

Percussion, if it is to be of any value, must be practised with the greatest care. The practitioner who has had but little experience in examining the chests of children will often err, even though he may be very skilful with adults. The difficulties which are liable to arise have already been set forth under the head of croupous pneumonia. It may suffice at this point to

call attention to the fact that vibrations of sound produced by percussion spread in every direction, and that in the chest of the child we have before us a comparatively very small mass, and at the same time one which is extremely susceptible of vibration. It is therefore necessary to employ a light, short stroke in practising percussion.

Diminished resonance indicates a diminished supply of air. If we fall back on this cardinal principle of Skoda's, it follows that diminished resonance can never determine whether atelectasis or pneumonic infiltration is the condition present. The matter of determining the presence of larger infiltrations is also made difficult, because, as was stated before, these usually appear symmetrically on both sizes. The only differences in resonance which can be depended on are those between symmetrically located spots on the two sides. For it is only here that we shall find the conditions outside of the lung which influence resonance anything like identical. It is therefore of even more importance here than in croupous pneumonia to pass around the chest in successive zones of percussion.

During later stages of the disease dense infiltrations are met with, sometimes scattered through the entire lung, which would be evident even to a beginner.

Auscultation furnishes us with a valuable sign. Unfortunately, however, it is the same that is furnished by catarrh of the finer bronchi as well. I mean the fine mucous râle which is very similar to the crepitant râle, but differs from the latter in being heard during expiration as well as inspiration, and in not being quite so fine. This râle is evidently produced in the smaller bronchi; it may eventually be associated with a genuine crepitant râle, and my ear, at least, is unable distinctly to distinguish between these two closely allied phenomena of sound. It is likewise of very little practical value to be able to distinguish a slightly ringing quality sometimes modifying this râle. Those who have keen senses and much experience will sometimes recognize it. If the condition present is merely that of extensive atelectasis, the breathing heard over these spots will, as a rule, be very feeble. In case of pneumonic infiltration the greater or less degree to which the bronchi are loaded with secre-

tions may produce every variety of sound, from the ill-defined, feeble, vesicular murmur to the intensely rasping bronchial breathing which almost pains the ear. The only way to get light on the subject is by repeated examinations, especially after a severe fit of coughing. I believe every experienced physician will agree with me that *the physical diagnosis of an acute and not very extensive catarrhal pneumonia belongs among the most difficult tasks in this department of our science.*

Among the *exanthemata*, miliaria may be mentioned as being of not unfrequent occurrence. Herpes is extremely rare. In very cachectic individuals I have occasionally seen petechiæ. If the eruption of measles is still present, this will grow less distinct, on account of the general anæmia of the skin as soon as the disease in the lungs reaches a high grade. This circumstance has played an important part in establishing the doctrine of the metastasis of exanthemata that have been "driven in." In tedious cases of catarrhal pneumonia, furuncular affections of the skin and pustular ecthyma are not rare.

I can make no accurate statement with regard to the frequency with which light attacks of *albuminuria* complicate this disease, and would merely say that I have often found albumen present, but have very rarely encountered more serious disease of the kidney. When it did occur, it was always in the form of chronic nephritis, never in the acute hemorrhagic form.

Once, in a case of prolonged catarrhal pneumonia, gradually passing into phthisis, which occurred in a young and very cachectic girl, I observed *noma vulvæ*.

The *brain symptoms* met with in catarrhal pneumonia vary greatly in intensity. Sometimes the only one encountered for a long time is a certain confusion of the intellectual faculties, which is now and then temporarily interrupted by a period of mild excitement. In other cases a picture is presented which corresponds exactly, even to its details, with that which characterizes tubercular meningitis. Both show a state of apathy, boring of the occiput into the pillow, slight muscular twitchings, vomiting, finally strabismus and violent convulsions. The only symptom which is perhaps less sharply defined in the cases now under consideration is the irregularity of the pulse, its intermissions

and variable fulness; but this, too, is not alike in different subjects. I consider it as downright impossible to determine with certainty, in any given case, whether a child suffering from catarrhal pneumonia has, at the same time, a moderate crop of tubercles in the brain or not. The last epidemic of measles which I saw at Tübingen gave me some additional data on this point. At the polyclinic there, post-mortem examinations are never refused. All the autopsies were made by my honored colleague, Schüppel, who is, beyond question, thoroughly competent to pronounce on the question of tubercles. It appeared, then, that the most violent brain symptoms might occur without any trace of tubercles, and again an abundant deposit of tubercles might be found in the brain of a patient who had presented no evidences of functional disturbance of this organ worth mentioning. The closest observation failed to reveal to me any truly reliable diagnostic tests. Even ophthalmoscopic examination, when it gives a negative result, is by no means to be taken as positive evidence against the existence of tuberculosis. For tubercles of the choroid are far from being constantly present in milder cases of tuberculosis.

The impossibility of drawing a sharp distinction, in this case, is evident upon a closer study of the anatomical changes which are common to the two conditions, and of the injuries to which in both instances the brain is exposed. Œdema of the brain, irregular distribution of blood in this generally anæmic organ, the influence of an abnormally elevated temperature, and the overloading of the blood with carbonic acid, all these are common to both affections. Qualitative differences can only be brought about by a localized tubercular inflammation affecting certain nerves. Quantitative variations may be produced by a change in any one of the injurious factors mentioned above, and such a change may occur at any moment in catarrhal pneumonia, whether associated with tuberculosis or not.

Complications and Sequelæ.

Bronchitis is the essential prerequisite of catarrhal pneumonia. We therefore always find them associated when this

form of pneumonia is present. I have, throughout this discussion, so strongly insisted on the significance of capillary bronchitis, that it seems unnecessary to dwell on it any longer.

Pleurisy without effusion is almost always present; with effusion it is very rare, and is to be regarded as a complication, that is, as a manifestation which does not necessarily follow the conditions produced by the pneumonia. In that case some other auxiliary cause must exist. What is it? We do not know. In the few cases observed by me the exudation was highly fibrinous or purulent. The slower forms of pneumonia seem more liable than the acute ones to be complicated by pleurisy with effusion. All that was said under the head of croupous pneumonia, concerning the diagnosis of this condition, holds good here. Only there are even greater difficulties in the way in the latter than in the former disease, and these are still further increased by the narrowness of the thorax in children.

In some epidemics of measles, *stenosis of the larynx* is found comparatively often in connection with catarrhal pneumonia. According to my experience the pneumonia usually precedes the attack of stenosis. The symptoms of the latter are the same as in other cases. In addition to a continuous, moderate degree of labored inspiration, every breath of which is accompanied by loud, rasping sounds, there are paroxysmal attacks of severe dyspnœa. Aphonia is always present, though it is often possible, by a strong effort of the will, to produce a momentary vocal sound. Expiration is either entirely free or but very slightly embarrassed.

In the great majority of cases the condition presented is that of a laryngitis, which, in addition to certain catarrhal affections, involves paralysis of the muscles that open the glottis. Unfortunately I have not been able to make laryngoscopic examinations in these cases, as the arrangements of the polyclinic prevented my doing so. I can therefore support my views only by indirect evidence. I would first call attention to the comparatively slight degree of stenosis which exists, except during the actual paroxysms. If there had been any considerable degree of swelling, this could not have been so, especially as the subjects were very young children, who, therefore, had

but a narrow larynx. Another very striking feature was that the attacks, no matter how severe, ceased as if by magic the moment the children were put into a warm bath. Emetics had no effect on the severity of the attack, or, at least, very little. This fact, which was very often verified, would be absolutely inexplicable if the stenosis depended on a permanent mechanical obstruction.

Finally, I adduce the favorable results reached. In spite of the most severe and prolonged attacks, I did not lose a single one of the seven children in whom I saw the higher grades of laryngitis during the recent epidemic of measles. Nor was I obliged to perform tracheotomy. I think that, taken as a whole, all these circumstances suffice for the support of my views. I would further state that at least two weeks, and more frequently four weeks elapsed before these attacks of laryngitis entirely subsided. Relapses were frequent, as were also the greatest variations in the gravity of symptoms from one day to another. Hoarseness usually persisted for some time after the close of the laryngitis, but I have never seen it remain permanent. I am rather inclined to account for the paroxysms, as Biermer [1] does in an altogether different set of cases, on the theory of a cramp of the muscles closing the glottis, which would need to be but very slight as long as their antagonists were paralyzed. Aside from this form of the disease, which is unaccompanied with danger, there is another one which is dangerous. And here we must distinguish between two sets of occurrences according to the order of time in which they follow one another: diphtheria following catarrhal pneumonia and catarrhal pneumonia following diphtheria. The latter does not concern us just now. Bartels [2] has set forth the leading points of this question in a very lucid manner. A pseudo-membranous laryngitis may however follow an attack of catarrhal pneumonia, which was itself the result of measles or whooping-cough. I use this unobjectionable term (pseudo-membranous) because it does not seem to me to be as yet positively determined whether the identity of the anatomi-

[1] Ueber Bronchialasthma. Volkmann's Sammlung, Bd. I., p. 54.

[2] Ueber die häutige Bräune. Deutsches Archiv f. klin. Medicin, Bd. II., p. 367 et seq.

cal conditions found in croup and diphtheria implies a similar etiological identity between these two affections. These cases of pseudo-membranous laryngitis often terminate fatally. At the same time it hardly seems to me as if a patient suffering from measles could offer a very good soil for the reception of true diphtheritic poison. I am unable, however, to adduce reliable data in support of this opinion, based on general impressions, and the few facts which I might recall from memory prove nothing.

If a person attacked with catarrhal pneumonia is predisposed to *tuberculosis*, there is danger that this tendency will be further developed by the disease. This danger is still more imminent if the tubercular disease has plainly manifested itself at some previous period, and only been temporarily dormant, and the risk is still further enhanced if the tubercular disease has been localized in the lungs.

This statement corresponds with our views of tuberculosis, so far as they rest on facts. Experience proves that the circumstances attending different epidemics will cause great variations in the frequency of this complication. Conditions of time and place may both make themselves felt, and sometimes the one may be evenly balanced, sometimes overbalanced by the other. I have good evidence of this from personal experience. Tubercular disease is incomparably more frequent in Kiel than in Tübingen, and yet, during the epidemic in the former place, Bartels found only three cases of tuberculosis in sixteen post-mortems, while in Tübingen I found four tubercular cases among nine deaths from catarrhal pneumonia. Of course these figures are too small to prove much. But they do show how much chance may have to do with the matter. If it just happens that a large number of tubercular candidates has accumulated, then an epidemic of measles or of pertussis will clear them out.

There is no doubt that *croupous pneumonia* may occur in connection with the catarrhal form. But in many cases it is so difficult to arrive at a diagnosis, even in the dead-house, that the microscope has to be appealed to for a decision.

Pneumothorax is a condition I have never met with, although Steffen[1] reports two cases complicating catarrhal pneumonia. Diseases of the *pericardium* are certainly very rare complications, and those of the *endocardium* still more so.

[1] L. c., p. 321.

The various forms of *chronic pneumonia* must be classed among the sequels, including the varieties characterized by contraction of the lungs as well as those associated with ulcerative changes.

The same is true with regard to *bronchiectasia* and *emphysema*. Complete or partial *pleuritic adhesions* are very frequent. Sometimes the development of acute tuberculosis after catarrhal pneumonia is delayed so long that it might be called a sequel rather than a complication. In young children the occurrence of a catarrhal pneumonia of any considerable degree of severity always acts unfavorably on their subsequent development. Often this is all that can be seen. At other times, however, *rachitis* is suddenly developed, without there having been any indication of it beforehand. Or such an extreme degree of general debility and cachexia may remain that any subsequent cause of disease terminates the young life. *Intestinal disease*, especially follicular intestinal catarrh, often follows, and may be accounted for as shown above.

After catarrhal pneumonia the *lungs*, as a general rule, are *very vulnerable*. This is proved by their constant tendency to catarrh upon taking cold, and by the fact that these catarrhs are usually widespread and obstinate. At the same time it is not common to find catarrhal pneumonia occurring repeatedly in the same individual. Diseases of the *heart*, compensatory hypertrophies, etc., as well as *amyloid degeneration* of the larger glands, are to be regarded as sequels of the sequels, and need not engage our attention at this time.

Diagnosis.

Let us consider, first, those points which can be made positively available in diagnosis. They are:—

1st. The demonstration of the existence of catarrh of the finer bronchi or of the operation of some cause which is so powerful an excitor of inflammation that we must suppose it to have produced inflammation either of a general character, extending throughout the pulmonary tract (as from the inhalation of chlorine, ammonia, etc.), or of more circumscribed extent (as by a foreign body in the bronchus).

If such a capillary bronchitis has preceded it, catarrhal pneumonia may follow,—not necessarily, however, inasmuch as other forms of pneumonia are possible.

2d. A consolidation of the lung, beginning at its base, arising slowly, at first not betraying itself by any prominent signs, and being usually bilateral. The breathing sounds at these points are very changeable, and gradually assume a bronchial character, being at first mixed with a peculiar, fine, mucous râle, very similar to a crepitant râle, but also heard during expiration. This sound assumes an increasingly ringing character, which is at first not well marked. The pectoral fremitus varies; when distinct consolidation occurs, it is, as a rule, greatly increased. *On inspection every inspiration is seen to be accompanied by a retraction of the lower ribs.*

3d. Aggravation of the general symptoms, such as dyspnœa, frequency of the pulse, fever, and other disturbances dependent on faulty respiration; the appearance and gradual development of the evidences of carbonic-acid poisoning; likewise changes in the character of the coughing fits.

4th. During the further progress of the disease, the non-typical character of the fever, the indefinite limitation of the disease, the fluctuations that occur in both general and local symptoms, and the absence of any critical periods.

On the whole, then, it will appear that the presence of primary infiltrations—that is, such as are not preceded by collapse,—can scarcely ever be demonstrated with certainty. This is simply due to the fact that the amount of space occupied by them is small, and that therefore they are neither accessible to physical examination nor do they produce any considerable general disturbance. In this case the local and general disturbances stand in a very different relation to one another from what they do in croupous pneumonia, because the general symptoms depend on and are caused by the local process. We are therefore never able, in a case of extensive capillary bronchitis, either confidently to assert or to deny the existence of such minute, pneumonic infiltrations. As a general rule, we are furnished with a demonstrable initial lesion in the form of collapse of the lung, from which, then, catarrhal pneumonia is developed more or less

rapidly. But the collapse alone is sufficient to explain the changes in the physical signs as well as those in the general condition dependent on defective lung-action.

I cannot, like Ziemssen, attribute any striking significance or value, as regards differential diagnosis, to the *character of the fever*. According to Ziemssen, the fever of capillary bronchitis never raises the temperature above 104° F. I have frequently observed the contrary; even in simple—not very extensive—catarrh of the bronchi, in little children, I have often seen a far higher temperature maintained temporarily. I was even able to verify some of these cases by the results of post-mortem examination. Neither do I believe that the thermometer is capable of establishing the distinction between collapse and pneumonia; for frequently collapse is nothing else than the result of increased fever, which produces superficial respiration.

In my opinion the *differential diagnosis between collapse and catarrhal pneumonia* can be determined only in two ways.

1st. The *diminution in volume of a certain portion of the lung, if distinctly demonstrated, is evidence of simple collapse; an increase in volume is evidence of infiltration.* If no alteration in the boundaries of the lung can be demonstrated, we must look for other evidence, as it is easy to imagine compensatory combinations that might prevent the former. Ziemssen has set this forth very plainly. We may obtain some help from the fact that resonance on percussion is usually less flat over collapsed than over infiltrated portions; that it has a slightly tympanitic quality over the former, and that, as a rule, phenomena of consonance are wanting. To all this, however, we must attach only a subsidiary value.

2d. *The question is settled by any therapeutic measure which succeeds in re-establishing the entrance of air into a portion of the lung that was devoid of air.* But nothing less than a positive result can be accepted in evidence. It is never possible, within a few hours, to clear up a section of lung that has been subject to pneumonic infiltration, and not always one that has been subject to collapse. The only positive demonstration, then, consists in a successful distention of the part.

It will be found very difficult, and perhaps for a time impos-

sible, to draw *the distinction between a case of catarrhal and one of croupous penumonia*, if one does not see the case until consolidation is complete. Valuable aid may be obtained from the history of the case. If this fails, then the question must be decided by the course of the disease. It is hardly necessary to recapitulate the points to be observed.

Even a slight pleuritic exudation may embarrass the diagnosis, if no marked evidences of displacement by pressure exist. What adds to the difficulty is, that catarrhal pneumonia is usually bilateral and situated in the lower lobes. Therefore the resistance against the pressure of an effusion is very considerable, and the heart is seldom crowded out of its place. The younger the subjects, and consequently the narrower the space within the chest for accommodation of these simultaneous processes, the more difficult will it be to distinguish them. Sometimes it is impossible. Here, too, I refrain from repeating rules which are generally known.

Inasmuch as in the large majority of cases *acute miliary tuberculosis* begins with an abundant development of tubercles in the lungs and with a high grade of bronchial catarrh, the differential diagnosis between a catarrhal pneumonia developed from an acute bronchitis and one developed from a tubercular bronchitis must depend chiefly on the history and on the course of the disease before the occurrence of consolidation. If the latter is already established, I generally decline to give a positive opinion, unless some positive data from a previous period are at hand. Among such data I should place evidence of the existence of a high grade of fever before the appearance of any local pulmonary symptoms. Possibly the condition of the retina may settle the diagnosis. The younger the patient the greater are the chances of error. In fact, a man is always on slippery ground unless he has had a chance to watch the course of the disease from the beginning. The significance of brain symptoms has been treated of above.

Whenever groups of tubercles in the lungs are associated with catarrh of the finer bronchi, it is very difficult to distinguish them from catarrhal pneumonia. The difficulty is still further increased if they are complicated with chronic pneu-

monic processes in the lower part of the lungs. The same thing is true of chronic pneumonia without tubercules. One will only be able to arrive at a probable diagnosis, unless he is familiar with the case from former examinations, or unless the fully developed physical signs of consolidation are inconsistent with the duration of the disease. Any conditions of contraction of the thorax may be of great significance; likewise any compensatory changes that are demonstrable in the heart.

Duration; Terminations; Prognosis.

It has already been stated that the *duration of catarrhal pneumonia is subject to the greatest variations.* The minimum to be allowed for the most acute forms, such as the pneumonia of measles, is about fourteen days. Ziemssen and Krabler[1] report such a case, in which, at the end of fourteen days, "nothing anomalous could any longer be found in the respiratory organs." It is hard to say whether in this case there was complete restitution to a state of integrity, as small, deep-seated accumulations are impossible to be demonstrated. Nor is their statement of a return to the normal temperature to be accepted as conclusive, because the temperature was only taken once a day, and that at evening. I have repeatedly found the evening temperature to be normal, or even subnormal, while during the morning or at noon it was considerably higher. So rapid a disappearance of the local signs would always lead me to suspect extensive collapse rather than firm infiltration. This does not imply that small infiltrations may not be found along with the collapse; indeed they are very likely to be. According to my own experience I should be inclined to allow *from one and a half to two weeks for the acute stage of catarrhal pneumonia, and at least an equal time for the period of restitution.* Chronic catarrhal pneumonia lasts for months. It is difficult to fix the limits of this disease, because very often its sequelæ are so gradually developed that it is impossible to say where they began. In catarrhal pneumonia *death* may ensue within the first few days; indeed, in the acute form this is not an uncommon thing.

[1] L. c., p. 136, Case 60.

Bartels cites instances of quite young children who died within the first twenty-four hours, and I have seen the same thing. In that case the disease had not passed the stage of collapse. In other cases the fatal result occurs later—though still during the acute stage—with all the evidences of carbonic-acid poisoning. If death is postponed until the disease has become chronic, it may be preceded by an acute exacerbation. Usually, however, the degree of marasmus is so great that it may be regarded as the cause of death. Slight aggravation of any one of the symptoms may then be enough to extinguish the flickering light.

The *rate of mortality* is far higher in catarrhal than in croupous pneumonia. The danger to life is very great.

Number of patients.	Number of deaths.	Authority.
68	29	*Bartels*, Virchow's Archiv, Bd. 21.
61	48	*Barrier*, Traité pratique des maladies de l'enfance, T. I., p. 258. Paris, Chamerot, 1861.
98	36	*Ziemssen*, Pleuritis und Pneumonie im Kindesalter, u. s. w., p. 329.
32	9	*Pfeilsticker*, Beiträge zur Pathologie der Masern. Tübingen, Fues, 1863, p. 115.
66	35	*Steffen*, Klinik der Kinderkrankheiten, u. s. w., Bd. I., p. 342.
325	157	*Per cent. of mortality*, 48.3.

In the above table I have combined the figures given by a number of reliable authors, which abundantly demonstrate the danger of catarrhal pneumonia. Those writers whose experience is chiefly in the children's hospitals in large cities make even a worse report. In the Foundling Hospital in Paris, according to Valleix,[1] out of 128 newly born children that were attacked, 127

[1] Cited by *Steffen*, l. c. I have been unable to find the original. This statement is not to be found in Valleix's text-book.

died. Steiner,[1] as the result of his experience in Prague, announces a mortality of two-thirds.

The table comprises catarrhal pneumonia in general, without reference to the nature of the antecedent disease. Ziemssen has already declared that the danger is less in the pneumonia of measles, where the attack is acute, than in those forms where it is insidious and slow. Undoubtedly he is right. Among 149 cases of pneumonia in measles, only 54, or 37.1 per cent., terminated fatally.[2] From the above data we may arrive at the following prognostic propositions:

1. *The danger to life in catarrhal pneumonia is in inverse ratio to the acuteness of the attack.*

The *age of the patient* is also an important factor. Bartels lost all children under one year of age, and after that period only a little over one-third. Ziemssen lost just one-half of the children under one year of age; from 1 to 3 years old, about two-fifths (17 out of 45); in the later years, only one-fourth (8 out of 31). Some mistake must have crept into Steffen's figures, as he reports a larger number of deaths than of patients during the first year. At all events, we may say that

2. *Before the age of puberty, the danger from catarrhal pneumonia grows greater in proportion to the youth of the individual.*

Inasmuch as catarrhal pneumonia is a local disease, the danger increases with the extent of the topical affection. Hence,

3. *In catarrhal pneumonia the danger is in direct proportion to the extent of the pneumonic process.*

The *powers of resistance of the patient* constitute an important element in every prolonged febrile disease. This is particularly true here, where, owing to the fact that the nutrient blood itself is chemically altered by reason of insufficient ventilation, and to the profoundly altered conditions of the circulation, the process of nutrition must be materially interfered with throughout the organism. Therefore, in children that were originally feeble, or that have become so, the disease runs a much more

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[1] Compendium der Kinderkrankheiten, p. 164. Leipzig, Vogel, 1873, II. Aufl.

[2] I obtained these figures from the sources given in the table.

pernicious course than in those that are strong. *Rachitic and scrofulous children, or those who have recently undergone any severe acute infection, are liable to far greater danger.* The same is true of *very fat* children, as the volume of blood in their case is less in proportion to the weight of the body than in that of well-nourished individuals who are not fat (Subbotin). Therefore,

4. *A diminution of the powers of resistance on the part of a person attacked with catarrhal pneumonia increases the danger to life.*

Among the single features of the disease which are especially unfavorable are smallness of the pulse, increased frequency of respiration, cessation of cough, great cyanosis, and loss of memory, as well as more prominent brain symptoms.

TREATMENT.

The study of the symptoms, as a whole, should lead us to *a definite conception as to what constitutes the essential danger to life in catarrhal pneumonia.*

Here, too, as in the case of croupous pneumonia, we find a twofold disturbance—*interference with the function of the lungs* and *fever*. But the rate of mortality indicates that in this case there must be something more going on than in the croupous form. In the latter no special danger is found to attend the earlier years of life, but rather the contrary. The height reached by the fever is about the same in both diseases. If we wish to make a fair comparison, we must select for this purpose the acutest forms of catarrhal pneumonia. In the latter, then, the fever differs only in being of longer duration ; and it is especially to be remembered that those attacked with catarrhal pneumonia have already been suffering from fever for a longer or shorter period preceding the attack.

An analysis of the changes produced in the lungs by catarrhal pneumonia and the conditions that precede it will reveal a far more serious *disturbance of the functions of the organ* than is usually found in croupous pneumonia.

The essential difference no doubt depends on the varied

developments of the bronchial catarrh. A wide-spread bronchitis, which leads to a diminution of the calibre of the tubes through which air is admitted—and this is accomplished by swelling of the mucous membrane—must result in an increase of the obstacles both to inspiration and to expiration. At the same time the area of contact between the blood and air is diminished, as less air enters the alveoli. Even supposing that the propulsive powers remain the same, the secretion poured forth by the inflamed mucous membrane affords an additional obstacle, the significance of which is increased by the partial destruction of the ciliated epithelium, which is itself one of the agencies employed in keeping the air-passages clear.

All these obstacles within the respiratory apparatus, caused by the catarrh, can be overcome only by an increase of vital force, which is to be supplied by the respiratory muscles alone. From the first moment of catarrhal invasion of the air-passages, therefore, an augmented task is imposed upon the respiratory powers. If the respiration becomes superficial, even for a short time, so that the complete expansion of the lung is prevented, then collapse results in the manner indicated above.[1] *The occurrence of this collapse offers of itself a new and serious obstacle, which I will call obstruction by adhesion* (Verklebungswiderstand), *and at the same time positively and completely shuts out a certain portion of lung-surface from all interchange of gases. This again adds to the task imposed upon the respiratory muscles*, provided that the same amount of air is to pass through the lungs in a given time. Another temporary relaxation of the activity of these muscles results in a new collapse, especially as the production of the latter is greatly facilitated by the existence of former collapses.[2] And thus the dread circle grows ever narrower. *The moment that collapse of the lung is developed, almost all that was said in connection with croupous pneumonia, with regard to overlaxing of the heart, holds true.* I especially insist on this point, that, under these circumstances, the same amount of blood that used to be

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[1] See page 204 and following.

[2] Compare the detailed description p. 206 and following.

conveyed to the lungs, within a given time, can no longer be conveyed thither within an equal time, except by an increase of the heart's action.

It still remains for us to inquire *how this temporary relaxation of activity in the respiratory muscles takes place;* or, perhaps more correctly stated, *why they are sometimes unable to effect such an expansion of the lungs as would prevent collapse.* Two circumstances are especially prominent: *the severity and extent of the catarrhal swelling, and the fever.*

If a growth in the intensity or the extent of catarrhal disease increases the obstacles interposed, if even very transitory accumulations of mucus take place within a bronchus, preventing the entrance of air, we may already have the conditions present that are capable of accounting for an interference with the full development of muscular activity. An equal degree of importance at least is to be attributed to the influence of fever on the respiration. The fact is thoroughly established that an increase of the temperature of the body causes more rapid and superficial respiration. There is no need that these variations in temperature should be very great. It is even possible that the mere normal rise of a moderate fever from morning to evening may suffice; an elevation of temperature to 102.5°, which is the least we have in every capillary bronchitis, might be enough to favor the supervention of collapse. There are causes enough apparent which might temporarily prevent the complete expansion of the lung. The rapid development of the process in measles, and its slower progress in pertussis, are explained by what has been said. The milder fever and the severe paroxysms of coughing are less favorable to the production of extensive collapse.

Another serious source of injury to the individual lies in the diminished pulmonary area that is open to respiration and the difficulty which the blood experiences in gaining access to that area. This affects the entire organism, and particularly those organs which have the most work to do, viz., the respiratory muscles and the heart, likewise the nervous system throughout its entire extent. A diminution of oxygen and increase of carbonic acid in the blood; then, on account of the

incomplete filling of the left ventricle, insufficient pressure in the renal artery; this is the constantly growing danger which necessarily results from the mechanical disturbances in the lungs. For not only is the collapsed or infiltrated portion of the lung impermeable to air, not only does the blood which is to be driven through these portions meet with greater resistance; the inevitable œdemas, the emphysemas, which are found in every autopsy, also cause a diminished interchange of gases and increased action of the heart. The *poverty of oxygen in the blood* causes a diminished working power in all the muscles as well as in those portions of the nervous apparatus which excite and regulate their activity. The *overloading of the blood with carbonic acid* produces a partial paralysis, a narcotism of the brain—perhaps of the entire nervous system. Persons who die of catarrhal pneumonia perish through insufficiency of the respiration. This may be developed rapidly or slowly, but it is essentially the same process whether in the acute pneumonia of measles or in the more chronic form which follows whooping-cough.

Aside from death through impeded respiration, we may also have it occur through *insufficiency of the heart's action.* This alone, however, is very rarely responsible for the fatal termination; in fact it never is, unless after unwise therapeutic interference, whether in the form of the abstraction of heat, the abstraction of blood, or the employment of remedies that weaken the heart's action.

Just as in croupous pneumonia I emphatically attributed to cardiac insufficiency the first place in the rank of fatal agencies, without thereby underrating the importance of impeded respiration, so, in catarrhal pneumonia, I believe I am right and safe in reversing this order, and announcing respiratory insufficiency as the principal cause of death. Of course it is to be understood we do not thereby ignore disturbances of the cardiac functions. For those injuries to the heart which exist in croupous pneumonia likewise obtain in the catarrhal form; only here insufficiency of the respiratory function predominates.

Throughout this entire discussion I have made no radical distinction between infiltrated and collapsed portions of lung. I

believe this is right, for there is scarcely any difference in their action as regards interference with the function of respiration. At all events, the change from atelectasis to pneumonia could only influence the results in a quantitative, not in a qualitative manner. An increased rise of temperature, longer duration of the disease, perhaps also locally some greater disturbance of the circulation,—this, and this only, might characterize a fully developed infiltration as compared to atelectasis.

I should like to advance two additional points in support of the views here presented. One is that children are exposed to so much greater danger during the earlier years of life that, before the development of actual pneumonic processes, they die very easily, presenting just the same picture as older persons, with fully developed infiltration. The second is actually merely the reverse of this. Strong men die of catarrhal pneumonia only when the most powerful hindrances, such as croupous or diphtheritic membranes, obstruct their bronchial tubes.

I have already intimated that sometimes, in the protracted forms of catarrhal pneumonia, *death may be caused by marasmus*. No doubt the ultimate cause is to be found in the gradual disintegration of the blood, combined with the deleterious effects of protracted fever. In the present state of our knowledge on this subject, it is evidently useless to attempt to go further into details. I need hardly state that the therapeutic indications would be different in the chronic forms.

The *prophylactic treatment* of catarrhal pneumonia naturally arranges itself under *two heads*, viz. : *prophylaxis against catarrh itself* and *prophylaxis against catarrhal pneumonia when catarrh is already present.*

The details of the first are more specifically set forth in another portion of this cyclopædia, and I shall therefore confine myself here to laying down general principles. The best protection against catarrhs, as against taking cold generally, consists in an intelligent treatment of the skin. Both extremes are to be avoided; children should not be coddled on the one hand, nor made the victims of a fanatical hardening process on the other. I advise people who are so situated that it can possibly be done, to give the children a lukewarm bath (at 86° F.) in the morning, and to follow this by pouring a bucket of cold water over them. The temperature of the latter may gradually be brought down

to about 55° or 60° F. I have them begin this at the end of the first year. In children as young as that the amount of water used for showering should, of course, be much less. The age at which these daily warm baths may be discontinued will depend somewhat on the circumstances of the family. They are then to be replaced by daily washing of the entire body at morning, or, better yet, with simple showering in an empty tub. If children are made to wear flannel next to the skin, and especially if they are not allowed to go without some sort of woollen underclothing in summer, all requisite precautions will have been employed. It may be necessary now and then to warn people against some undue exposure of the surface of the body in following the dictates of fashion, or against wrapping the little ones up like Esquimaux when the weather grows cooler. But people soon learn themselves to take a wise middle course.

When a child is seized with a severe catarrh accompanied by fever, then the main problem for the physician is the prevention of pulmonary collapse.

The first thing to be considered is the outside surroundings. The importance of the admission of good, pure air has already been insisted on elsewhere. In these cases it is so absolutely essential, that, in case of need, it is better to smash a window-pane than to put up with a stench. A draft is better than bad air. The higher the fever the more need of air. The inhalation of quite cool air often excites coughing; therefore it is usually unpleasant to the patients, and because the cough is followed by pain, they usually think that the catarrh is also worse. After an extensive experience of over twelve years in the practice of the polyclinic, I may say that I have never seen this opinion confirmed. I think the truth is about as follows:

In the *acute stage* of catarrhal pneumonia, as it arises, for instance, in cases of measles, the temperature of the air is a matter of very little consequence; the lower temperature is perhaps the more favorable as it is more likely to induce cough, and thereby to favor the expansion of collapsed portions of lung. In the stage of convalescence, as well as in those cases of pneumonia which run a slow course from the beginning, cold air is perhaps objectionable, as severe and frequent paroxysms of

coughing may lead to the aspiration of the secretion from the bronchi into the alveoli, and thus, besides causing the formation of disseminated pneumonic infiltrations, favor the development of emphysema and bronchiectasis. Special attention should be given to the temperature of the air in whooping-cough. But in all the conditions last named, if the choice is between cold and good air on the one hand and warm and bad air on the other, experience teaches that the patient who chooses the cold, good air fares the best. Perhaps the explanation of this fact lies in the circumstance that the dust with which the stagnant air of an inhabited room is charged, containing as it does organic and inorganic substances, includes among them no inconsiderable number of such as are capable of exciting inflammation.

When everything can be arranged to order, I usually direct the maintenance, in the sick-room, of a constant temperature of from 59° to 64° F.

A considerable degree of *moisture in the air* seems to me to be very desirable in the more severe catarrhal affections of the bronchi. If the atmosphere surrounding the patient contains much vapor of water, it follows, as a physical necessity, that the exhalation of water from the lungs will be diminished. It is evident then, that the bronchial secretion will grow less tenacious, that it will offer less resistance, and be more easily coughed up. The best and easiest method of surrounding the patient with the requisite amount of steam is this: Let a close screen be set at a suitable distance around the bed, and let the space between the screen and the wall of the room be roofed in with bedclothes, thus constituting a hut, one side of which should be formed of a movable curtain. Let such openings be made near the bottom of the screen as will admit the nozzles of one or more tea-kettles, which should be set outside, over spirit-lamps. By heating these kettles, the requisite amount of steam can be generated, and the curtain enables one to regulate the moisture as well as the warmth of the patient's circumscribed apartment, while at the same time a sufficient change of air can be secured. This arrangement, by which the patient is not obliged to retain any special position, but can sleep or wake as suits him, is certainly preferable to any attempts at attaining

this end by atomizers of any kind. I know of cases where one single tube from a boiler, led into the room, furnished steam enough for the whole room. Where this is practicable, it is the best way. One further advantage to the patient from this uniform moist atmosphere is, that it encourages a constant fluxion to the skin and perspiration. This is no slight advantage. It is certainly possible to abort a catarrh outright in this way, if it is not too old.

The medicines ordinarily used in these cases are *muriate of ammonia* and *carbonate of soda.* I consider their action in influencing the amount of the secretion or the character of the mucus as very problematical. *Oil of turpentine* certainly is capable of diminishing the quantity of the bronchial secretion. In the slower forms of catarrhal pneumonia I have therefore made extensive use of this remedy. I give about five drops, three or four times a day in milk. I have no experience with regard to its use in acute cases. *The problem of diminishing swelling of the mucous membrane, and limiting its secretion will certainly never be solved by giving medicine.*

Hence the necessity for an increase from time to time in the amount of vital force to be supplied by the respiratory muscles in order to accomplish the adequate expansion of the lungs. In order to effect this most fully, the anatomical structure of the muscles must be disturbed as little as possible, while, at the same time, their nutrition must be maintained to a degree proportionate with the demands made upon them. *Fever*, damaging the heart and respiratory muscles as it does, is our greatest and most obstinate enemy. In further evidence of this truth I may refer to what was said on the subject in connection with croupous pneumonia. In the present instance special importance is further to be attached to superficial respiration, which accompanies every rise of temperature, as a circumstance favoring collapse of the lung.

It is possible that powerful respiratory movements may now and then carry a substance capable of exciting inflammation into some portion of the lung that was before merely collapsed, and that the development of a catarrhal-pneumonic accumulation is thus favored. I accept this possibility; and still the indication for interference, in the direction already set forth, seems to me unequivocal.

Both objects—the expansion of the lungs and the combating of fever—may be best accomplished by the use of baths of a moderate temperature followed by cold affusions. In children the temperature of the water may be higher than in the case of adults, because in the former the surface of the body which is exposed to the abstraction of temperature is larger in proportion to its weight. In old and weak persons, where this condition does not exist, there are manifest physiological reasons bearing in the same direction. Exceptionally, in case of very high temperature and very obstinate fever, cold baths must be employed in catarrhal pneumonia. All that has been said before on this subject applies here also.

I usually order baths of the temperature of from 77° to 86° Fahr., and have the patient remain in the bath for twenty or twenty-five minutes. When he comes out of it, from ten to twenty quarts of water are to be poured over him from a moderate height. This water may be reduced to a few degrees above the freezing-point. The affusion must be rapid, and so managed that the most thorough showering of the neck, back, and breast is effected. This procedure will act even more energetically if the patient is placed in such position as to subject the entire surface of the body to the influence of the cold, which may be accomplished by having the child held, in a standing posture, in the bath-tub. The abstraction of heat effected by such an affusion is comparatively slight. If it is desirable, however, to avoid even this, deep respirations may be secured in another way. *If a stream of water, not more than a third of an inch in thickness, is directed against the back of the head, over the region of the medulla oblongata, a spot will soon be found, the irrigation of which produces the most violent respiratory efforts.* I discovered this accidentally, on my own person, and have since frequently verified it at the sick-bed. Deep inspirations can be induced in this way, even when quite a high grade of carbonic-acid poisoning is present.

This method is, therefore, to be employed even in the last stages of catarrhal pneumonia.

During the *acute stage*, bathing must be regulated according to the temperature of the body, and the vigor of the medical

treatment must depend on the energy of the respiratory muscles. It may be stated, as a general rule, that the younger and weaker the patient is, the greater the need of active interference, and that the sooner this interference is begun the less vigorous will it have to be. I am, therefore, much more careful in younger than in somewhat older children, to treat every fever associated with anything like a considerable catarrh by means of the general abstraction of heat. For I believe that we may thus directly prevent the development of collapse and catarrhal pneumonia.

On the whole, there is no reason for deviating from this plan when *consolidations* are found present. I have repeatedly and most thoroughly satisfied myself that *a few hours after an affusion, spots which were formerly dull became resonant again.* Hence I cannot doubt that this therapeutic measure fulfils a *causal indication* in these cases.

The *cold packs*, which have been so much used, are more severe, if thoroughly carried out, and at the same time not as effectual as the method of bathing I have recommended. Furthermore, they are accompanied with great discomfort to the patient, and consequently are but seldom used with sufficient energy by the attendants. I only have recourse to them when I can do no better.

The *diminution of cough*, which so soon attracts attention during the development of catarrhal pneumonia, may call for additional therapeutic aid. Vigorous fits of coughing usually attend and follow the cold affusions, but they are not always sufficient to dislodge the bronchial secretion. I am fond of prescribing decoction of senega, with eighty minims of anisated spirit of ammonia,[1] to be given every hour or two as soon as the cough seems to be insufficient. Coughing begins immediately after swallowing the medicine, as any one may convince himself at the bedside.

If extensive mucous râles and the increasing dyspnœa indicate the presence, within the bronchi, of a large amount of mucus which cannot be otherwise expelled, then we must have

[1] Oil of anise, 1 part; alcohol, 24 parts; water of ammonia, 5 parts.—*German Pharmacopœia.*

recourse to *emetics*. But it should never be forgotten that these can only give temporary relief. A certain degree of collapse of the heart is associated with the act of vomiting itself. If the substance used as an emetic remains in the stomach long enough to be absorbed, it must, after passing into the circulation, exert a more or less paralyzing influence on the heart. Hence the rule that an emetic should be allowed to remain in the stomach as short a time as possible. As *apomorphine* produces collapse only when given in very large doses, this is decidedly the best agent to use in these cases. The hypodermic administration of the same makes the physician independent of the co-operation of his patient, and explosive vomiting follows quite promptly. I have employed apomorphine very frequently, and am very well satisfied with its action. But I would not recommend it as a preparation of universal applicability, because it is not always possible to obtain it pure, and it soon undergoes decomposition. These are very serious objections to its use in country practice, for impure or decomposed preparations act on the heart. The best way to keep it is in a solution of about ten grains of the hydrochlorate of apomorphine to a fluid ounce of glycerine and water. One Pravaz syringeful should be injected.

I *never give tartar emetic in divided doses*, but, even in the case of little children, administer a full dose of three-quarters of a grain, with from eight to fifteen grains of powdered ipecac. A part of this dose will be spilled anyhow in giving it, and the more quickly and thoroughly the act of vomiting follows, the smaller is the probability that any of the drug will remain in the stomach. Under some circumstances it may be necessary to administer a second dose before seeing any result. This, however, will be very rare, unless the emetic has been postponed too long. In well-marked narcosis its administration should be preceded by a cold affusion.

The use of *quinine* as an antipyretic, of *wine* as an article of diet, or of the *cardiac stimulants*, should be subject to the same rules as in croupous pneumonia. We cannot, from the nature of things, expect the same striking results from the use of the cardiac stimulants here as in the croupous form.

In *protracted cases* especial attention is to be given to the

matter of *nutrition*. Wine is most strongly to be recommended as a means of preserving the strength. *Nestle's Food* ["Nestle's Mother's Milk?"] is strongly to be recommended, in the case of younger children, as a substitute for the breast. As it can be given in concentrated form it may even be preferred to the latter for somewhat older children.

Let us cast a glance at the *older methods*. *Venesection* would hardly be recommended nowadays by any half-way intelligent physician. Ziemssen [1] did good service towards banishing this method from children's practice when he supported his argument against it by a case in which a child had been thus killed *secundem artem*. This seems to have been effectual, it is to be hoped, forever. But little better than the foregoing is the practice, still, alas, too common, of treating severe bronchitis and catarrhal pneumonia by means of *small quantities of tartar emetic dissolved in an infusion of digitalis*, to which are added large *blisters*, *irritating ointments*, and other means of torture. The spread of the disease is directly favored by such methods, inasmuch as the heart and respiratory muscles suffer downright injury. *Mercury*, venerated, according to the old-time custom, as an "antiplastic," is to be met with everywhere in children's practice. In catarrhal pneumonia it is used by inunction, in the form of mercurial ointment, and given internally as calomel. In moderate doses it does less harm than the treatment mentioned above. But there is no shadow of evidence in favor of its exerting any beneficial action, unless we are willing blindly to accept as such the mere dictum of "authorities."

But enough. If any one wishes to go further into this question he will find it treated of in a comprehensive manner by Köhler.[2]

The treatment of the *sequelæ* and *complications* may be disposed of in a few words.

I wish especially to speak of the measures to be adopted in that form of *laryngitis* which has already been described as complicating pneumonia. The tumultuous onset of the disease is very likely to drive one to vigorous treatment, to tracheotomy

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[1] L. c., p. 264.

[2] L. c., p. 762 et seq.

as well as to the previous persistent use of emetics. In these severe cases of dyspnœa I have employed nothing but warm baths—somewhat above the temperature of the body—of long duration, and have never lost a child under this treatment. Once I was prepared for tracheotomy, because there was the possibility of the existence of a severe local affection, but I did not have to operate. We must never forget that, after opening of the larynx, children are no longer able to cough satisfactorily, as, in the absence of closure of the glottis, the expiratory compression of the chest is insufficient. Therefore we should wait as long as we safely can. On the other hand, it is true that stenosis of the larynx very seriously interferes with the mechanical relations of respiration within the lungs. It particularly results in the undue accumulation of blood and air in various portions of the lung—a condition of things on which Bartels lays great emphasis.[1] But in the form of stenosis to which I refer, this consideration is less prominent, because the narrowing of the glottis becomes excessive only in paroxysms, and these paroxysms can be modified in other ways.

The application of the *constant current* to the larynx was tried by me in one case. It seemed to effect some improvement, although of a very transitory character.

Ziemssen is right in advising a *residence* in the country after recovery from pneumonia. I would further add, that attention to the development of the respiratory muscles is most urgently to be advised. Every one who has gone through an attack of catarrhal pneumonia is more exposed than before to the danger of becoming phthisical, and the best prophylactic against this is the development of powerful pectoral muscles.

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[1] Deutsches Archiv f. klin. Medicin, l. c.

HYPOSTATIC PROCESSES IN THE LUNGS.

Piorry, Traité de médecine pratique, Tom. IV. Paris, Baillière, 1843.

Hypostatic pneumonia and hypostatic conditions of the lungs were first recognized as a distinct form of pulmonary disease through the labors of French writers. Pre-eminent among them is Piorry, who handles the subject with great clearness, and whose teachings are based upon a rich experience. He likewise gave the disease its name.

It was at first believed that the anatomical changes now recognized as hypostatic, took place *during the death-struggle*, or *even after death*. This view was based upon the circumstance that the localization of this condition in the lungs was so evidently influenced by the force of gravitation. Piorry proved, by experiment, that a hypostatic condition diagnosticated during life did not alter its location after death, under the laws of gravitation. He caused the bodies of those in whom, during life, he had recognized hypostasis of the posterior portion of the lung, to be laid on their bellies after death, and left so for from twenty-four to thirty hours. On opening them the hypostasis was still found on the posterior surface ; the force of gravitation had not been enough to influence the blood which had once been displaced. As Piorry had made his diagnosis long before death, it was evident that this condition did not result during the death-struggle.

By means of these experiments hypostasis ceased to be a condition of but little pathological significance. If it occurred during life, then it must have an influence on life.

PATHOGENESIS AND ETIOLOGY.

Two conditions must be fulfilled for the production of pulmonary hypostasis :

1st. *Diminution in the propelling power of the heart.*

2d. *The prolonged maintenance of the body in one position.*

The force of gravitation acts so powerfully in that portion of the lung which lies lowest, that the enfeebled heart's action is not capable of driving the blood through it with sufficient rapidity. The result of this, at first, is a mere retarding of the circulation. But if the unfavorable conditions continue, it ends in stasis. When the walls of the blood-vessels are no longer adequately nourished by the coagulating blood, they refuse longer to retain their contents, which then escape both in the form of plasma and of corpuscles. This discovery of Cohnheim's has made it easy to understand the pathogenesis of hypostatic conditions of the lung.

The *extent* of hypostatic infiltration, in any given case, will depend on the intensity of the conditions producing it. The closure of a certain number of branches of the pulmonary artery must necessarily increase the resistance to the flow of blood through the lungs, as a whole. The occurrence of hypostasis, therefore, must of itself constitute a circumstance favorable to the spread of the process. In fact, it is not altogether uncommon to find the posterior aspect of both lungs the seat of hypostatic infiltration.

Everything that embarrasses the full expansion of the lungs, through the respiratory muscles, must be considered as a *favoring cause:* such are, *meteorism*, *deformities of the thorax*, *old pleuritic adhesions*, etc. For the power which, in addition to that of the heart's action, serves to secure the movement of the blood through the lungs, is supplied by the respiratory muscles (force of aspiration). If the latter partially fails, as it does in imperfect expansion of the lungs, then the heart must either exert a greater power or the amount of work previously done cannot be accomplished. Piorry insists that hypostatic conditions are *more frequent and more extensive on the right side* than on the left, and attributes this partly to the presence of the liver on that side. The convexity of the spinal column towards the right, in the lumbar region, and the greater weight of the extremities on this side, are also adduced as causes. The basis of the whole deduction lies in the fact that whatever prevents

the expansion of the lung increases the obstacles to the circulation of blood. Although Piorry probably goes too far in claiming that *the diseases of the right side are always more intense than those of the left*, yet, on the whole, there is doubtless much to be said in justification of his views. It will be remembered that in a previous portion of this treatise[1] it was stated that the first evidence of pseudo-crepitation accompanying the simple pulmonary collapse of persons with superficial breathing occurs at the posterior and inferior portion of the right lung. Another strong argument is found in the fact that, if circumstances more unfavorable to the expansion of the left lung are developed, through rachitis for instance, then hypostatic infiltration occurs on the left side. The difference between the right and left sides of course only appears if the patient has lain continuously on his back. If, for any reason, he habitually lies on one side, the laws of gravitation will assert themselves too powerfully to permit these smaller anatomical predispositions to appear. *The most dependent portion of the lung is the one in which these hypostatic changes begin.* This is a proposition which must be accepted as absolute.

Probably every physician has had occasion to see instances of such hypostatic changes on one side. I recently met one in the case of a typhus patient who had thrombosis of the femoral vein. The leg was laid in a box, and the patient turned somewhat on to his left side. The hypostasis occupied a narrow lateral section of the lung, corresponding exactly to that portion which lay most dependent. The posterior sections were quite free.

Does an *inflammation of the lung* actually exist—is the term "hypostatic pneumonia" correct? Here, too, we must agree with Piorry, who answered this question in the negative in his nomenclature,[2] and afterwards still further confirmed this opinion. If, however, no inflammation is present at first, it is also true, on the other hand, that this condition furnishes the occasion for the development of inflammation. This occasion must be characterized as directly favorable thereto. The inflammatory infiltra-

[1] Compare page 83.

[2] He calls this form of disease "Pneumonémie hypostatique," and gives as a synonyme, "Engouement pulmonaire."

tions resulting therefrom, both lobar and lobular, are always catarrhal.[1]

The *conditions which give occasion to the rise of hypostatic infiltrations of the lungs*, so far as they can be designated by name, are varied in nature. But they all fulfil the requirements which we have found to be essential.

All *acute infectious diseases*, associated with severe and continued fever, which produce insensibility and are not treated by the antipyretic method, may be placed within this category. An insensible typhus patient, who has slid down to the foot of the bed and lies there motionless, with feeble pulse and shallow respiration, furnishes a typical instance of the circumstances under which these hypostatic conditions arise.

It is easy to understand why the cold-water treatment should prevent this accident. It prevents insensibility, it strengthens the heart's action, it implies a frequent change in the position of the body in taking the baths, and it necessitates deeper respiration.

Acute articular rheumatism very often leads to hypostasis, even when the degree of fever is insignificant. In these cases the laws of gravitation have an opportunity to exert even more than their usual influence, on account of the absolutely unchanged position which the patient so often maintains. In acute rheumatic arthritis it is quite common to find the most extensive hypostasis lasting for months.

Cases in which the patient is obliged to maintain a uniform position in bed for a long time, on account of *wounds*, fall within the same category.

In *the aged* and *the cachectic* the relative importance of the causes concerned varies somewhat. Here paralysis of the heart's action ranks first. Therefore, in these cases, hypostatic conditions often result within the first few days of lying in bed.

The *development of genuine catarrhal inflammation* from hypostatic conditions is greatly favored by the pre-existence of a bronchial catarrh. The same is true of everything that may act as an irritant to the bronchial mucous membrane and thus produce catarrh.

[1] Compare *Rindfleisch*, Lehrb. d. path. Gewebelehre, III. Aufl., p. 362.

We therefore find that all severe diseases of the lungs following hypostatic infiltration are more liable to occur after acute infectious diseases associated with catarrh of the bronchi.

Pathological Anatomy.

In the lighter forms the parenchyma of the lung is quite unchanged. The lungs still appear to contain air, the vessels in the dependent posterior and inferior portions are distended with blood; on incision the lung is found somewhat moister than usual. A small amount of bloody, foamy fluid may perhaps be found within the bronchial tubes. By the gradual increase of all these changes a condition of the lung is finally reached which we designate as *splenization*. Then the parenchyma is of a deep blue or blackish-red color, and uniformly saturated with blood. If the hypostatic condition is recent the tissue is firmer, but crepitates more feebly than in health; it still, however, contains air and floats on the water.

In the highest grades of prolonged hypostatic pneumonia, the posterior portions of the lung, especially along their lower borders, will be found entirely devoid of air and somewhat collapsed. The parts containing no air are strikingly shrivelled and flaccid, the pleura covering them is uniformly dull, ecchymotic, and covered with a thin fibrinous coating which can easily be stripped off. On a cut surface the infiltrated spots appear of a uniform, dull, bluish-red color, and but very little thick blood oozes from them. "The blood seems to have melted away, as well as the tissue" (Rokitansky). The walls of the alveoli and of the interstitial tissue are swollen, the alveolar septa are enlarged, the alveoli are narrowed and filled with the extravasated components of the blood and swelled alveolar epithelium.

Within the *bronchi* a bloody, mucous—or, more commonly, a bloody purulent—fluid will be found. The mucous membrane of the bronchi is of a diffuse, dark-red color. *Serous effusions* into the pleural cavity are frequent.

This picture may be greatly varied by the presence of widely extended catarrhal pneumonic infiltrations at a later stage of development.

It is not necessary to go into details again, and it will suffice to refer the reader to what has been said in connection with catarrhal pneumonia. The complication of the latter with hypostasis eventually presents all those manifestations which have been noticed under the head of the non-complicated form.

Symptomatology and Diagnosis.

The occurrence of hypostasis at first causes no considerable disturbance, either of the general functions or of those peculiar to the respiratory apparatus. This is due to the gradual development of the condition, to the presence of other severe maladies at the same time, or to the extreme old age of the individual. The pathological conditions betray themselves first by somewhat increased frequency of breathing, slight cyanosis, and an inconsiderable acceleration of the heart's action. The pulse becomes more rapid, but the artery is not as well filled, and is therefore easily compressible. Piorry calls attention to the fact that in old people, at the beginning of the disease, the mouth sometimes instinctively remains open during sleep, as if to secure breath more easily. Then the tongue is found dry in the morning, while the lips and teeth are coated with thickened mucus. Piorry says that he was often led to make a local examination by observing this sign. In *higher grades* of the disease, evidences of impeded circulation of the blood and defective interchange of gases become more prominent. The veins of the neck swell, the face grows somewhat dusky, and the right ventricle is said to lie in contact with the walls of the chest throughout quite a space. The liver is enlarged, and œdema of the lower extremities supervenes. There may be no cough whatever. If there is, it is likely to be accompanied by the expectoration of small quantities of muco-purulent sputa, which rarely contain blood. These symptoms, gradually increasing in intensity, may grow to the fully developed picture of carbonic-acid poisoning, followed by death; or the respiration gradually grows freer, the signs of impeded circulation and of imperfect aeration of the blood recede, and the disease takes a favorable course. Febrile movement is by no means always present. It may be conjectured that *elevations of*

temperature are due, not to the hypostatic conditions as such, but to a catarrhal pneumonia which has supervened. The fever, when it is not to be attributed to a previous disease, is always irregular, and seldom accompanied by any great rise of temperature.

The *local symptoms* of hypostasis *demonstrable by physical examination* are the following:

At first, diminished resonance on percussion, beginning at the lower angle of the scapula, and on auscultation a lessening, sometimes almost a cessation, of the respiratory murmur, which is vesicular, or may be quite indefinite in character. At the point of attack the vocal fremitus is weak. If hypostasis is complicated with a coexistent catarrh, new features, foreign to the former disease, will appear. Mucous râles, for example, are usually absent in simple hypostasis. The dulness on percussion and the auscultatory signs, as a rule, extend slowly *from below upwards*. There is a period at which absolutely no breathing is to be heard over the consolidated portion (Piorry). Then mucous râles gradually become audible, those in the larger tubes appearing first. In case of a fatal termination, extensive œdema of the lungs supervenes, accompanied by the auscultatory signs peculiar to that condition. No great difference is perceptible if the catarrh results in pneumonic consolidations. For, even where no true pneumonia exists, intense bronchial breathing with sonorous râles may be recognized over the consolidated portions of lung.

In one case of extensive hypostasis on both sides, in a patient suffering from acute articular rheumatism, I found the acoustic signs, for weeks at a time, to be precisely the same as in croupous pneumonia. There could not, however, have been any extensive pneumonic consolidation, for the patient was free from fever, and all evidences of consolidation disappeared within a week after she left her bed.

It appears from such not very infrequent clinical observations that hypostasis, when not complicated with catarrhal pneumonia, may end in complete resolution.

If widespread catarrhal pneumonia is present, a very multiform picture of pulmonary disease may be presented, the essential features of which, however, will still be those of catarrhal

pneumonia. The reader is therefore referred for particulars to the sections devoted to that disease.

In a malady so completely under the control of individual circumstances, it is likewise impossible to lay down any general rules with regard to *duration* or *prognosis*. If the conditions producing the disease are manifestly within reach of treatment, or if they are such as will spontaneously cease after a time, then the prognosis, in simple hypostatic affections, is usually good. Instances of this sort are to be found in those forms of the affection occurring with acute rheumatic arthritis, or with simple wounds. In other cases more weight will have to be attached to the fact that hypostasis, as a necessary result, produces disturbances of the pulmonary and cardiac functions. The value of these disturbances, as threatening life, will then have to be taken into account, in addition to the other pathological conditions present. The existence of catarrhal pneumonia as a complication is always most undesirable, and affects the prognosis unfavorably in proportion to the extent of the infiltration.

TREATMENT.

As hypostasis owes its origin to mechanical causes, the therapeutic indications, in theory at least, are very simple. We should endeavor:

1st. *To prevent the force of gravitation from acting continuously in any one direction.*

If there is reason to fear the development of hypostasis, care should be taken that the patient frequently changes his position. He should be made to lie now on his right side, and again on his left, be propped up with pillows, and not be allowed to remain lying on his back long. If circumstances permit, he should even be induced to lie upon his belly for a time. Sometimes, however, all this postural treatment is absolutely contraindicated on account of the severe pain produced on movement of any kind.

2d. *The heart's action must be stimulated.*

Under some circumstances it may be necessary to have recourse directly to cardiac stimulants. At other times digitalis

may serve the purpose, as in case of cardiac insufficiency with very rapid but feeble contractions—a condition often encountered among the aged. If the grade of fever is quite high, antipyretic treatment may be indicated. Very often the only means that can be adopted to accomplish the end in view is the promotion of nutrition by an appropriate diet. During convalescence from severe febrile affections this is generally our main dependence.

3d. *The respiratory muscles must be made to act with energy.*

I am fond of causing those patients who are capable of the effort systematically to take deep inspirations, calling into play the auxiliary respiratory muscles. The individual should be made to sit upright, seize some point of support with his hands, and take a given number of deep inspirations to the minute. This may be repeated every two hours, or less frequently, according to the strength of the patient. If fever still persists, the subject thereof may be put in a bath, which must be only a few degrees cooler than his body at the time; at the close of the bath, the duration of which will be determined by the state of the individual, affusion with somewhat cooler water is employed.

The physician must determine, in any given case, which of these proceedings is applicable. In an affection depending so entirely on individual influences, we can only sketch the most general therapeutic outlines. This may be said in closing, that *the sooner a patient is enabled to leave his bed and walk about, the more rapidly will hypostasis disappear.*

PNEUMONIA FROM EMBOLISM.

Cohn, Klinik der embolischen Gefässkrankheiten. Berlin, Hirschwald, 1860.—*Virchow*, Gesammelte Abhandlungen zur wissenschaftlichen Medicin. Hamm, Grote, 1862, II. Auflage.—*Panum*, Experimentelle Untersuchungen zur Physiologie und Therapie der Embolie, Transfusion und Blutmenge. Berlin, Reimer, 1864. Sep.-Abdr. aus Virchow's Arch., Bd. 27/29.—*Cohnheim*, Untersuchungen über die embolischen Processe. Berlin, Hirschwald, 1872.

PNEUMONIA from embolism has no doubt always existed, but its recognition is one of the achievements of modern times. Those interested in the literature of embolism will find what seems to be an exact compilation on this subject in Cohn. The scope of the present work forbids my going into this at length.

The conditions presented by this disease were first thoroughly treated of in Laënnec's[1] work, the title of the chapter being "On Pulmonary Apoplexy." The anatomical description is clear and unequivocal; the description of symptoms is appropriate, all the main points being noticed. The fact that the pathogenesis of this affection escaped the ken of that great inquirer is to be explained by the profound obscurity which at that time involved all those facts upon which our modern teaching of embolism rests. These were brought to our knowledge first by the pioneer labors of Virchow, aided in many directions by the valuable additions of Panum, and finally by the conclusive observations of Cohnheim. All these authors paid special attention to embolism of the lungs in particular.

PATHOGENESIS AND ETIOLOGY.

Pneumonia from embolism arises through the migration of solid bodies into some of the ramifications of the pulmonary

[1] L. c., Bd. I.

artery. Where, then, do these bodies originate, and what is their character? We know of two sources, *the cavities of the right side of the heart* (perhaps, in rare exceptional cases, also those of the left side), and *the veins of the systemic circulation.* From either of these, solid bodies may be carried into the pulmonary artery.

The nature or composition of emboli divides them into two classes. The one acts in a purely *mechanical* manner, as nothing more nor less than a mere plug; the other, in addition to its mechanical action, possesses *specific* qualities, which may, in a general way, be designated as *infectious.*

The changes which may take place after the introduction of emboli into the lungs depend upon the above-named factors. Their extent will be governed by the size of the embolus, their nature by its nature. *Non-infectious plugs, if indeed they cause any structural changes, produce hemorrhagic infarction; infectious plugs result in embolic or metastatic abscesses.*

In the vast majority of cases, we shall succeed in finding an embolus in the artery leading to that division of the lung which shows hemorrhagic infarction. The occasional failure to find it may depend upon the difficulties presented to such a search, which, in the smaller branches of the pulmonary artery, are considerable. But it is also possible that the same anatomical condition—hemorrhage into the lung tissue—may be attributed to another cause, viz., to the simple rupture of enfeebled capillary vessels. Rokitansky, and Gerhardt, too, amongst clinicians, advocate the exclusively embolic origin of hemorrhagic infarction. Virchow, at least indirectly, has advised caution in the adoption of this view, and Cohnheim has very emphatically done the same. It seems to me that no conclusive arguments have yet been advanced in favor of the exclusively embolic origin of hemorrhagic infarction.

The fact that precisely the same changes which are found to follow certain pathological conditions may be produced by artificially inducing similar conditions (experiments of Virchow and Panum) proves that *where these pulmonary changes exist we should expect to find embolus.*

The point of origin of emboli is most frequently *the heart,*

and particularly its *right side.* If deficient force of the heart's action results in the formation of clots in the right auricle, this may be the starting-point of embolism of the pulmonary artery. Such a condition of things in the auricle is liable to arise as soon as valvular disease, which is not sufficiently compensated for, throws upon the heart a burden of insuperable obstacles. Gerhardt[1] has given special prominence to the right auricle as the starting-point of pulmonary embolism in persons suffering from cardiac disease. Indeed, a firm clot will very often be found here, intimately interwoven with the muscular framework. And it cannot for a moment be doubted that a prolonged stagnation of blood at this spot would first produce a disturbance in the nutrition of the endocardium, which, as soon as it was sufficiently advanced, would, in turn, bring about a gradual extension of the clot. But it would be a mistake to seek for the origin of pulmonary emboli in the right auricle alone, in persons having disease of the heart. There are cases enough in which a post-mortem shows nothing but death-clots in the right auricle, while yet considerable hemorrhagic infarctions are to be found.

In the post-mortems of four subjects of heart disease, having pulmonary infarctions that could be demonstrated as originating in embolism, I found thrombi in the right heart in only two cases (Tübinger Poliklinik of last year).

Those who believe that pulmonary emboli may originate in the left side of the heart, must suppose that the obstructing plugs pass through the capillaries of the general circulation before they become stranded in those of the pulmonary circulation. It must be admitted that this is, *à priori*, possible. Indeed, we find conditions presented on the dead body that probably do not admit of any other solution.

Pulmonary emboli are very often formed from clots which have originated in the veins of the general circulation and have thence found their way to the right side of the heart. These constitute a very frequent cause of hemorrhagic infarction in the subjects of heart-disease. Amongst the most frequent peripheral sources may be mentioned, all large external wounds, the

[1] Thrombosis cordis dextri. Würzburger medicin. Zeitschrift, Bd. V., p. 221 et seq., 1864.

venous thrombi of the uterus in puerperal women, those clots which have their origin in what is called marasmic thrombosis, bed-sores, ulcerations and suppurations of various kinds.

The size of the emboli is an essential factor in determining the extent of the pneumonia resulting from them. In case of hemorrhagic infarction, this is the only factor that comes into play, whereas in metastatic abscesses other influences make themselves felt. Infarctions are of variable size, sometimes embracing points no larger than a pin's head, at other times involving an entire lobe. Usually, however, they run from the size of a hazel-nut to that of a hen's egg.

The sudden closure of a main branch of the pulmonary artery usually ends in death so soon—producing the most violent dyspnœa and instant collapse—that there is no time for the occurrence of further structural changes. Sometimes, however, the closure takes place gradually, by the deposit of fibrine upon an embolus lodged within the pulmonary artery. Then those large infarctions are produced, involving an entire lobe. I lately saw a case in which the middle and lower lobes of the right lung were completely infarcted. The main arterial branch of the right lung was filled with a brownish-red, moderately firm thrombus. The patient was sixty-eight years old, and had cardiac disease. Embolism was caused by the escape of a clot from the right auricle.

In case of *non-infectious* plugs, the extent of the infarction depends solely upon the area supplied by the stream which is arrested through closure of the artery. Infarction only takes place in so far as this artery is a terminal artery (Endarterie) in the sense used by Cohnheim. In case of *infectious* emboli the situation is different. Here the intensity of the infecting agent makes itself felt, and the inflammation which follows may extend into a domain supplied by a different artery. Aside from the activity of the virus we must here take into account a factor, which varies in different individuals, viz., the power of resistance of the tissue attacked. This is, to say the least, a subordinate consideration in those cases where infarction depends on purely mechanical causes.

According to Cohnheim, it very rarely happens that an infected embolus produces hæmorrhagic infarction and metastatic abscess as well. Cohnheim adopts the view that the occurrence of an abscess, which implies a genuine inflammation, can-

not result from the mere closure of a terminal artery. Immediately after the closure, circulation ceases behind the plug, and thus the most essential condition for pus-producing inflammation is removed. He gives the following explanation for those cases where, aside from typical abscesses and infarctions, a peculiar mixture of the two is to be found in the lungs—peripheral collections, the outer portion of which answers to the characteristic image of infarction, while close to it is an equally well-pronounced collection of pus. He believes that a second and infectious embolus has lodged on the hither side of the point of obstruction; that this has produced the abscess, while the infarction depends on the purely mechanical action of the simple embolus in the terminal artery.

This explanation strikes me as whittling things down to rather too fine a point of dogmatism. I cannot see why an embolus saturated with ichor, when it has plugged up a terminal artery, may not communicate some irritant to the neighborhood, whether it be in the form of a soluble, diffusible substance, or of living organisms capable of exciting inflammation. In this way vascular tracts still open to circulation may, and in fact must be, reached, and react through inflammation. Under this view of the case, Cohnheim's main proposition still remains unassailed. He himself, in another place,[1] claims that the *intensity of the suppuration*, under some circumstances, depends on *peculiar* favoring circumstances, and he designates as such the intensity of the virus and the susceptibility of the organ infected.[2]

The processes undergone in the formation of an embolic abscess are, therefore, the same as take place in every genuine inflammation. It remains for us to consider the *method of development of hemorrhagic infarctions*. I here follow Cohnheim, whose views on this subject I regard as unassailable.

If an artery anywhere is closed by the entrance of an embolus, only one of two things can follow: either circulation is permanently arrested in the area supplied by the obstructed artery, or it is restored by means of collateral connections. In

[1] Embol. Processe, p. 110.

[2] Ibid , p. 109.

the latter case nothing is seriously disturbed: a supply-pipe failed, but its function was filled by others, and thus the original condition was restored again. A very different result follows in case of the arrest of an arterial stream, which cannot be equalized by means of the intervention of neighboring forces. By means of the pressure existing in the veins, which is a positive force over against the obstructed artery, the entire tract supplied by that artery becomes filled with blood (Anschoppung), always supposing that the veins have no valves. But the motionless mass of blood here accumulated is not capable of permanently nourishing the walls of the vessels. Thus, after a certain time, the formed elements of the blood, too, escape from the vessels whose walls are imperfectly nourished, and so we have hemorrhagic infiltration of the tissues (infarction). The anastomosis between different branches of the pulmonary artery is not sufficient to establish extensive collateral circulation, therefore we are very likely to meet with infarction in the lungs. But as there are no absolutely "terminal" arteries, there is a possibility that sufficient collateral circulation may be developed. "This is particularly true of all those branches of the pulmonary artery which are situated near the root of the lung or in the interior of the organ. As the arteries accompanying the bronchi in this situation are surrounded and enclosed on every side by the lobules and their alveoli, the number of the arterial anastomoses, which might serve for the equalizing of the interrupted circulation, must be almost unlimited. It is quite different in the case of an artery, which has only to supply a few peripheral lobules, for even if the small *lateral* anastomoses are not lost, those which otherwise lie directly *behind* the main artery concerned must be lacking. Under these circumstances it is very easy to develop conditions in which the arrangements for lateral anastomosis are not sufficient to effect rapid and complete equalization of the circulation; in other words, the branch of the pulmonary artery becomes practically a terminal artery" (Cohnheim).[1] Attention is further called to the fact that *the bronchial arteries cannot effectually come to the rescue*, as they only form capillary anastomoses with the pulmonary artery within the alveolar septa.

[1] Embol. Processe, p. 74.

In another place Cohnheim declares that *infarctions of the lungs are always located at the periphery of the organ.* This is certainly a necessary conclusion if one entirely accepts his account of the mechanical conditions within the pulmonary circulation. But this we cannot do. The only inevitable condition precedent to the rise of an infarction is the insufficient re-establishment of the circulation beyond the point of obstruction. This condition will also be fulfilled at the root of the lung, if anything interferes with the circulation there. Penzoldt,[1] who worked under Gerhardt, reports autopsies which prove that infarction may occur elsewhere than at the periphery. I have seen the same thing myself. The explanation given for it is merely an application of Cohnheim's fundamental law, and seems to me to be valid. If the circulation in the lungs has been materially interfered with by the occurrence of a number of peripheral infarctions, then the collateral circulation at the root may become insufficient, and an artery situated there may, to a certain extent, become a "terminal artery." An embolus entering that region under these circumstances may produce infarction.

The *shape of infarctions* is determined by the method of their production. Every artery subdivides by bifurcation, and advances from the root of the lung to its periphery, embracing an ever-increasing area within its domain. Hence it follows that an infarction corresponding to the bifurcation of the vessel advances, in the form of a wedge, to the surface of the lung, *the apex of the wedge being always directed toward the root of the lung, and the base towards its periphery* or pleural surface. *Abscesses*, on the contrary, are more apt to assume a *globular form;* even those resting against the pleura not touching the latter by a broad base, but only by a small segment of their globular surface (Cohnheim).

This corresponds with the tendency of all fluids to assume a spherical form. This will take place wherever the same amount of resistance is found on all sides—a condition which, in the case

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[1] Ueber den hämorrhagischen Infarkt der Lunge bei Herzkranken. Deutsch. Archiv f. klin. Medicin., Bd. XII., p. 13 et seq.

under consideration, may safely be assumed to exist in the lungs.

Pathological Anatomy.

On laying open an entirely fresh hemorrhagic infarction, the cut surface will be found to be of a dark blood-red color and moist; the lung tissue will contain no air, and, on moderate pressure, fluid blood without foam will escape.

If *some days* have elapsed since the occurrence of infarction, the latter will present itself as a hard, infiltrated mass, rising somewhat above the plane of the normal tissue, and usually peripheral in its situation. On transverse section, a firm, dark-red, wedge-shaped consolidation will appear, situated with its base to the surface and its apex towards the root of the lung, and often giving marked evidences of granulation. The single granules are, as a rule, larger than those of croupous pneumonia—a fact which was emphasized by Laënnec.[1] The tissue is uniformly firm, friable, and dry, throughout its entire extent. The fluid that oozes sparingly from the cut surface is blood, which is mingled with many minute black fragments of tissue.

An embolus is found obstructing the artery leading to the infarcted portion of lung.

A bloody and foamy, or merely a foamy, fluid is found in *the bronchi.* More rarely these are found filled with a fluid which is chiefly blood. The surrounding lung tissue is either unchanged, or, more frequently, œdematous—probably also hyperæmic for a certain distance. The pleura over the part affected is uniformly dull, sometimes highly vascular, and in rare instances inflamed and covered with a fibrinous coat. In large infarctions it is common to find a serous or bloody serous effusion into the pleural cavity—sometimes this is very considerable.

Microscopic examination shows that the increase in volume of the part affected is caused by blood. This blood is infiltrated throughout the entire lung; it is contained within its tissue, in the bronchi and in the alveoli. In the form of clots it fills the bronchi as well as the alveoli; hence the granular appearance

[1] L. c., p. 299.

of the surface when cut. *Embolic abscesses* are, anatomically, similar to common ones. They usually present a spherical collection of pus, surrounded by slightly hyperæmic, somewhat œdematous, but otherwise healthy lung tissue.

The anatomical changes undergone by an *infarction of long standing* are as follows:

The firm, dry, reddish-brown collection fades out and assumes a light yellow color. This depends on the death and fatty degeneration of the fibrine, as well as of the extravasated blood-corpuscles, which afterwards undergo absorption. The lung tissue itself generally remains intact, appears wasted and transformed into a relaxed structure, rich in connective tissue and moderately pigmented. Many infarctions are healed in this way.

Gerhardt[1] says that he has seen degeneration into a bloody, odorless fluid, containing hæmatine crystals and tissue débris.

Rokitansky[2] claims that the matters extravasated may become fluid again, and be partly absorbed and partly expelled through the bronchi. The parenchyma of the lung then gradually returns, according to him, to its normal condition.

Pulmonary gangrene constitutes a more rare method of termination. In that case it is supposed that there is compression of the nutrient vessel, the bronchial artery, and that gangrene is caused by exposure of the necrosed tissue to the influence of an atmosphere impregnated with materials capable of exciting this process.

An infarction cannot itself suppurate because no circulation takes place within it. But it is possible for an inflammatory action round about it to set up such a line of demarcation that it will be fenced off from the healthy tissue. It then resembles a foreign body within the lungs.

An infarction that has become gangrenous or a metastatic abscess may perforate into the bronchi, or into the pleural cavity. In the same way *perforation externally*, through the walls of the chest, occasionally takes place.

The action of the pleura demands special notice. Infectious

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[1] In Penzoldt, l. c., p. 17.

[2] L. c., p. 80.

emboli are usually associated with genuine pleuritis, the exudation of which may be sero-fibrinous, or it may be purulent. It is not necessary that it should be the latter.

In *simple infarctions* there is often an *effusion into the cavity of the pleura*. But this does not necessarily imply that such an effusion is an *exudation*, it may just as well be supposed to be a *simple transudation*.

Hopf[1] reports the post-mortem appearances in five cases. In four there was an effusion into both pleuræ, in one case nothing is said with regard to the presence of fluid. In *all cases* thrombi had entered only one lung, the other was free.

Penzoldt[2] has two observations that belong here. *In both there was embolism only of one side, but effusion on both sides.*

Four autopsies of mine during the past year resulted as follows:

No. 1. Extensive infarction, a main trunk of the pulmonary artery being closed *on the right side; effusion on the right side, the left pleural cavity free.*

No. 2. Infarctions *right and left;* effusion on the *right side alone*, but adhesions of the left pleural walls.

No. 3. Infarction on the *right*, an effusion *on the right* of more than a quart, and *on the left* of about three and a half fluid ounces.

No. 4. *Infarctions* on *both sides* and *effusion on both sides.*

All these cases were those of persons having heart disease. It is well known that in insufficiency of the heart serous transudations are common enough. The facts presented would seem to indicate that these effusions were more in the nature of a transudation than the result of inflammatory action.

Finally it may be mentioned that *the right lung is much more frequently the seat of infarction* than the left.

Symptomatology and Diagnosis.

Inasmuch as hemorrhagic infarction and metastatic abscess always arise as complications of some grave disease, it is impossible to draw a sharp, characteristic image of them alone. The question is rather, what symptoms, supervening on those of a previous disease, may lead us, with greater or less certainty, to suspect the existence of embolic pneumonia.

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[1] Zur Diagnose der hämorrhagischen Infarkte. Diss. Tübingen, 1865.

[2] L. c.

The first thing to be determined is, *whether the primary disease is such as may give rise to the formation of thrombi, which might subsequently act as emboli.*

These diseases have already been referred to under the head of Etiology. Marasmic thrombosis is often to be found in very remote portions of the venous tract, as in the pubic plexus, the sinuses of the brain, etc. (Penzoldt, Gerhardt), so that sometimes a very careful post-mortem examination will be required in order to find the original thrombus. It is probable that those cases in which the left side of the heart is supposed to be the source of a pulmonary embolism, are, to a great degree, to be explained on the ground of an insufficient post-mortem examination.

The closure of one of the smaller pulmonary branches usually produces no symptoms. We cannot determine at present how large an infarction must be in order to make itself recognized. Aside from all local manifestations, the gravity of the primary disease will make itself felt as an essential factor in the case. If the original disturbance is but slight, then the change in the aspect of the disease, caused by even a moderate-sized infarction, may stand out very prominently. This is not the case, however, where, for example, the primary affection is cardiac insufficiency in its last stage.

We should draw a distinction between the *general* and *local* symptoms.

One of the initial manifestations under the *first* head is an *increased frequency of respiration, which may advance to the most violent dyspnœa.* In case of the complete closure of one of the larger pulmonary branches, this dyspnœa will be very acute, and will arise suddenly without any warning, inasmuch as the entrance of blood into a large capillary area is suddenly arrested. The interchange of gases immediately stops within this area; the blood, as a whole, therefore, grows poorer in oxygen and richer in carbonic acid. Even with smaller infiltrations, a considerable amount of dyspnœa will be found at first. Here it is probably due less to the absolute extent of surface disqualified than to the fact that the lungs are already so greatly disturbed in their function; it is the last drop that overflows the bucket, already full to the brim. But however important and

essential this symptom, it is still well known to every man who has treated many patients with heart disease, in the last stage, how often they suffer from an increase of dyspnœa without any apparent cause. Where pulmonary embolism, therefore, is suspected in a person who has not heart disease, the existence of dyspnœa should be considered as of much greater diagnostic value.

It is hardly necessary to say that with the increased dyspnœa any cyanosis or evidences of impeded circulation that may be present will be aggravated.

A *febrile movement* may be set up, though not necessarily. There is so much uncertainty with regard to this, that the temperature cannot, in any sense, be regarded as a positive diagnostic sign. It is true that, as a rule, there is no considerable rise above the normal standard,—the elevations reaching about 102° Fahr. It is also true that the *fever does not generally rise until several days after the entrance of an embolus.* Penzoldt reports a case in which, three days after the supposed accident, the temperature, during twenty-four hours, ranged from 102° to 103.2° Fahr. I consider any attempts to fill the gap, in our knowledge on this subject, by experiments on the lower animals, as worse than useless. We know so little about the normal course of the temperature in these animals, that all deviations from the normal standard which are reported—this standard being generally nothing more than the temperature observed before the experiment—are utterly worthless.

Chills, whether occurring once or repeatedly, are also an inconstant symptom. They are of especially little value in pyæmic patients, as the frequent rigors to which they are subject cannot be attributed with certainty to the occurrence of embolism, much less to that of pulmonary embolism. I believe I may conclude, from my own observation, that, in simple infarctions, chills are as frequently absent as present.

Coming to the *local symptoms* we find some firmer points on which to base a diagnosis.

Prominent among these is the *quality of the sputa* in hemorrhagic infarction. The expectoration is from dark-red to black in color, and the blood which it contains is intimately mixed

with tenacious mucus. If any considerable amount is raised, it will be seen to contain comparatively little air. An expectoration of this character may follow very soon after the entrance of an embolus, or some time may elapse before it appears. A characteristic point is that *this expectoration continues for a considerable time;* it may often be observed for a period of eight or ten days.

The *amount* expectorated is ordinarily not very large From three and a half to six fluid drachms a day is usually all that is expelled.

Embolism is soon followed by *cough.* If a catarrhal affection of the bronchi has preceded the accident, of course nothing can be made of this symptom. Inasmuch as the catarrhal affection is very likely thus to have pre-existed, the only advantage of the cough is that it facilitates the expulsion of the characteristic sputa.

Pain, dependent on the pleural irritation which arises soon after the entrance of an embolus, is a very constant symptom of the presence of a foreign body closing a terminal artery and causing infarction. This pain presents the peculiarity characterizing pleurisy, viz., an aggravation in intensity on movement of the pleural surfaces.

Those signs which can be recognized by *physical exploration* appear only when a *more extensive* peripheral portion is the subject of infarction. Therefore, as a rule, they are absent in the more deeply seated embolic abscesses. An exception to this rule must be noted, if we may so far extend the scope of the term as to include effusion into the pleural cavity. This may perhaps be regarded as a secondary symptom, but it is a tolerably constant one.

If the infarction or abscess is *of sufficient size* and *situated at the periphery*, the physical symptoms of pulmonary consolidation will appear: dulness on percussion, the increase of pectoral fremitus, blowing respiration, with prolonged expiratory murmur.

All this is very easily laid down in theory, but it is usually very difficult, in practice, to establish these signs. The widespread pulmonary catarrh of persons with heart disease; the

difficulty of causing such sick patients to maintain the requisite position long enough for the exploration of such delicate points; then, furthermore, the existence of pleural effusion;—all these combine, at the bedside, to make a very difficult task out of what, to the *à priori* deductions of the writing-desk, seemed a sufficiently easy matter.

The following may be mentioned as circumstances giving support to the diagnosis of infarction in patients with heart disease: physical evidence of the acute extension of the area of cardiac dulness; the cessation of abnormal murmurs in the heart; irregularity of the heart's action, provided this did not exist before. These symptoms prove insufficiency of the heart, and perhaps the first may also be taken as evidence of thrombosis of the right heart. They are, therefore, only indirect and not positively diagnostic signs.

To sum up. *The diagnosis of hemorrhagic infarction* is, first of all, to be based on *etiological* grounds. *Pulmonary infarction is not to be thought of unless undoubted sources for the production of embolism can be demonstrated.* INFARCTION *may be recognized with tolerable certainty from the manifestations above described, taken as a whole, but never by any single symptom.* METASTATIC ABSCESS, *on the contrary, in the vast majority of cases, baffles all attempts at a strictly scientific diagnosis. It m y be suspected, but can rarely be recognized.*

Prognosis.

The prognosis always depends more upon the primary disease than upon the accident which we call pneumonia by embolism. Observation in the dead-house often shows cicatrices of pulmonary hemorrhagic infarction in the subjects of heart disease. Infarctions, therefore, may be healed. It is likewise true that embolic abscesses are not necessarily fatal to life. But in the vast majority of cases we have to deal with fatal conditions, which are accelerated by pulmonary embolism. It follows, then, that every embolic pneumonia, if at all extensive, has a tendency to shorten life.

The prognosis, therefore, is always doubtful—generally bad.

TREATMENT.

There are no fixed points in this affection on which to base direct therapeutic interference; for we have to deal with mechanical processes, which we can no more cause to recede than we can arrest the fall of a stone by our simple word of command.

It appears to me that Penzoldt utterly misapprehends the necessities of the case, when he attaches therapeutic significance to the occurrence of the effusion within the pleural sac, regarding the exudation as a compressory bandage applied by Mother Nature, "which prevents further extravasation and promotes absorption."[1] The escape of blood can only take place, anyhow, within the domain presided over by the obstructed artery. Pleuritic exudation, in so far as it does not depend on an infectious embolus, corresponds in bulk with the extent of the infarcted portion. The larger the extent of the peripheral region infiltrated with blood, the greater, *ceteris paribus*, will be the effusion. But this effusion takes place only *after* the development of the infarction. It compresses the entire lung, producing less effect on the portion filled with stagnant blood than on that less resisting portion containing air. The danger of infarction depends, in the first place, on the diminution of that surface where blood and air meet. What good, then, can be accomplished by a compressory bandage, which injures that portion of the tissue capable of performing its functions, and does no good to the part that is incapable of action? For I think that the very hypothetical advantage of its favoring reabsorption must be counterbalanced by the very positive injury to respiration.

The duty of the physician, then, consists in prolonging life in each individual case, until the *vis medicatrix naturæ* accomplishes the healing process. It will usually be necessary to strengthen the heart. Gerhardt[2] states that digitalis is generally not adapted to this purpose.

It is unnecessary to repeat, at this time, what was so fully set forth under the head of croupous pneumonia, with regard to the classification and the use of cardiac stimulants.

I likewise decline to enter into a discussion of those therapeutic measures which, while practised under the sanction of medical authority, do not appear to me to be based on scientific principles.

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[1] L. c., p. 30.

[2] L. c., p. 240.

HYPERÆMIA, ANÆMIA,

HÆMORRHAGES, ATELECTASIS, COLLAPSE, ATROPHY,

EMPHYSEMA, HYPERTROPHY, GANGRENE,

NEW-FORMATIONS,

AND

PARASITES.

HERTZ.

ANÆMIA, HYPERÆMIA, AND ŒDEMA OF THE LUNGS.

Hyperæmia and Œdema.—*Albertini* (Comment. de Bonon. sc. inst., Tom. I.), and *Barrière* (Observations anatomiques, 1753) alone of the earlier observers seem to have had a measurably correct appreciation of œdema of the lungs. We first obtain a true knowledge of this condition from the excellent description of its anatomy and symptoms by *Laënnec*, who traced back its pathological lesions to organic diseases of the heart, to certain affections of the lungs, and to a serous diathesis, and distinguished it from the serous transudation which occurs during the agony of death.—*Laënnec*, Traité de l'auscultation médiate, 1819.—*Hodgkin*, Lect. on morb. anat., II., p. 127.—*Andral*, Anatom. pathol., II., p. 135, and Précis d'anatomie pathologique. Paris, 1829.—*Piorry*, Clin. méd. de l'hop., 1838.—*Fournet*, Rech. clin. sur l'auscultation, 1839, I., pp. 280, 283, u. 302.—*Williams*, Diseases of the Lungs. London, 1828.—*Mendelssohn*, Archiv f. physiolog. Heilkunde, 1845, IV., p. 2.—The same, Der mechanismus der Respiration und Circulation oder das explicirte Wesen der Lungenhyperämie. Berlin, 1845.—*Woillez*, Archiv gêner. de Médecine, 1854, Mai. Schmidt's Jahrbücher, Bd. 83, p. 355.—*Köstlin*, Archiv f. physiol. Heilkunde, XIII., 1854, p. 2.—*Oppolzer*, Allg. Wien. med. Zeitung, 1860, Nr. 36 u. 38.—*Rokitansky*, Lehrb. der pathol. Anat., 1861, III.—*Hermann*, Pflüger's Archiv f. Physiologie, III., Jahrg. 1870, p. 8.

Capillary Ectasis.—*Andral*, Précis d'anat. path., 1829.—*Hasse*, Anatomische Beschreibung der Kr'kh'ten der Circulations- und Respirationsorgane. Leipzig, 1841, p. 293.—The same, Pathol. Anat., I., p. 293.—*Rokitansky*, Lehrbuch, 2. Aufl., 1842, III., p. 57; 3. Aufl., 1861, III., p. 45.—*Scoda*, Abhandlung üb. Ausc. u. Perc., 1844, p. 269.—*Virchow* in his Archiv, 1847, Bd. I., p. 461.—*Dittrich*, Beiträge, z. pathol. Anatomie der Lungenkrkhten. Erlangen, 1850. Prag. Vierteljahrschr. 1851, Jahrg. VIII., Heft 3.—*Heschl*, Compendium der pathol. Anatomie. Wien, 1855, p. 380.—*Isambert* et *Robin*, Mémoire sur l'induration, etc. Compt. rend. des séanc., Ser. II., Tom. II. Paris, 1856 Gaz. de Paris, 1855, 29-31. Schmidt's Jahrb., Jahrg. 88, p. 177.—*Friedreich*, Virchow's Archiv, Bd. X., p. 201.—*Bamberger*, Lehrbuch der Krankheiten des Herzens. Wien, 1857, p. 204.—*Buhl*, Virchow's Archiv, Bd. XVI., p. 559, with an Appen-

dix by Virchow.—*Zenker*, Beiträge zur normalen u. pathol. Anatomie der Lungen. Dresden, 1862.—*Buhl*, Virchow's Archiv, Bd. XXV.—*Rindfleisch*, Pathol. Gewebelehre, 1–3. Aufl.—*Colberg*, Deutsches Archiv f. kl. Med., Bd. II.

ANÆMIA OF THE LUNGS.

Anœmia of the lungs may be caused by any very considerable *loss of blood*. Thus it may follow the wounding and opening of any large vascular trunk, hemorrhages from the nose, lungs, stomach, intestines, or severe metrorrhagia; it may be due to *exhaustion of the blood* by acute and chronic diseases (typhus, tuberculosis, cancer, etc.); to a *diminished production of blood* (chlorosis); to *loss of the fluids* of the body and to general *marasmus*; and likewise to *thickening of the blood* in consequence of great loss of its serum (as in cholera, chronic diarrhœa of infants, extensive burns, etc.). As a rule, atrophied, inflated, emphysematous, and compressed lungs are likewise found in a bloodless condition.

An anæmic lung is dry, flaccid, collapsed, and has no turgidity or resistance. In children it is of a whitish-red, in adults of a grayish-white color, and more or less abundantly speckled with points of black pigment. The mucous membrane of the bronchi is also pale and anæmic. Occasionally also in adults an intensely bright red condition of certain portions of the lung is observed, which was considered by Stokes to be due to inflammatory changes. Rokitansky opposed this explanation, and proved that there are healthy portions of lung in which respiration has persisted after the arrest of the circulation during the final agony. Hence, the air penetrating the lungs comes repeatedly into contact with the coagulated blood in the capillaries, and the abundant supply of oxygen produces the bright, fiery red color. Furthermore, the distribution of the blood in an anæmic lung is sometimes irregular, so that the anterior portions of the organ contain less than the posterior.

In anæmia of the lungs characteristic symptoms are wanting. They coincide with those of general anæmia. If any special symptom can be ascribed to the lungs, it is the *dyspnœa* which now and then accompanies an excessive degree of anæmia.

The existence of this affection can only be conjectured after consideration of the etiological factors and the characteristic symptoms of general anæmia, and after all it is of no practical importance, for it requires no special treatment which is not already indicated by the general anæmia or by the diseases which have given rise to the latter.

HYPERÆMIA AND ŒDEMA OF THE LUNGS.

Etiology.

Hyperæmia of the lungs is caused either by an increased determination of blood to these organs—*active hyperæmia, hyperæmic fluxion, pulmonary congestion*—or in consequence of diminution or retardation of the flow of blood from them—*passive* or *obstructive hyperæmia.*

Pulmonary congestion results primarily from an increase of the arterial pressure, which may be due to increased action of the heart, or to very extensive contraction of the arteries either of the periphery of the body, or of the abdominal viscera, or of individual parts of the lungs themselves.

Increased action of the heart is most frequently observed about the age of puberty, and thereafter until the twenty-fourth and twenty-fifth years of age, in those individuals who have grown rapidly, and have a long, narrow chest, thin skin, slight development of the thoracic muscles and wide intercostal spaces. In such persons trifling causes, which produce a slight increase of the heart's action, are often sufficient to determine a congestion of the lungs. This may occur after any violent bodily exertion, which other persons could endure with impunity, such as running, dancing, riding, going up stairs, or climbing a hill; further, after moderate exercise in a cold atmosphere, mental excitement, public speaking, reading aloud, singing, and also after the moderate use of warm and spirituous drinks. These same exciting causes may, when carried to an extreme, produce pulmonary congestion by increased action of the heart, even when no special predisposition thereto exists, such as is produced by general debility, or by a slight excitability, a so-called

erethism, of the heart. Thus it may be caused by violent bodily exertion, especially during great heat, by excessive use of strong liquors, and, according to Niemeyer, by the psychical excitement of insanity and delirium tremens, especially when the patients have to be controlled by force. Like Lebert, I have been unable to convince myself of the etiological influence ascribed to this last agent, although I have frequently had the opportunity to observe the use of the so-called strait-jacket. The reason why increased action of the heart causes hyperæmia of the lungs only, and not a uniform distribution of the more forcibly propelled blood over the whole body, is because the capillaries of the pulmonary air-cells, being for the most part uncovered and exposed, are, during each inspiration, surrounded by rarefied air, and do not at the same time receive from the neighboring tissue the necessary compensating pressure from without. Under such circumstances a special increase of the blood-pressure takes place in these capillaries, which become greatly dilated, and hyperæmia ensues.

I do not think that erethism of the heart can always be considered the sole active agent in the production of pulmonary congestion in those individuals having narrow chests, slight muscular development, and thin skins. In such persons there are undoubtedly other conditions influencing the pulmonary circulation, which must be taken into account. One of these, although it has not been proved anatomically, is relaxation of the walls of the capillaries, which may be inherited and congenital, or caused by constitutional anomalies, or by a variety of other diseases such as rachitis, scrofula, chlorosis, etc. Further, the respiratory act seems to be not unimportant. Insufficient expansion of the chest, due to feebleness of the thoracic muscles, impedes the circulation, and causes retardation and accumulation of blood in the inadequately distended lungs.

Physicians and laity have long been aware of the ill effects upon the heated body of *cold drinks*. Aside from the many chronic affections which may be developed from this cause, and which do not concern us at present, active congestion of the lungs may thereby be induced, a fact which clinical observations as well as physiological experiments seem to prove.

On a very hot August day in Greifswald, a number of workmen were engaged carrying turf for a brewer, and one of them, who was about twenty-eight years old, thought he would cool the water by placing it upon the ice in the cellar, where the barrels of beer were cooled. This man, otherwise of a tolerably strong build, had a moderately kyphotic chest. After drinking freely of the ice-cold water, while perspiring, he was suddenly taken with a sensation of great distress, and fell to the ground. I was immediately summoned, and found him with congested face, fixed eyes, wild expression, and much dyspnœa, while coarse, moist râles could be heard even at a little distance; at the same time he was coughing, and from time to time expectorated very frothy and bright-red sputa. His pulse was frequent and pretty full. From what I learned from the other laborers and from the symptoms, I had no doubt that the case was one of acute congestive hyperæmia of the lungs, and proceeded to bleed freely at once. While the blood was still flowing, the man began to recover; the congestion of the face subsided, the respiration became freer, the pulse slower, the râles less marked, and in the course of half an hour he was feeling much relieved. By means of rest, a horizontal position, diet, and a pretty brisk purge, he was enabled to go about again without any difficulty a day or two afterwards.

The experiments in this direction, made by Hermann and Ganz upon dogs and cats, explain very satisfactorily the symptoms which occur in man.

One or more syringefuls (capacity about 55 ctm.) of water, at a temperature of 32° Fahr., were injected through an œsophagus tube into the stomachs of animals that had been stupefied with morphia and deprived of motion by means of curare, and whose carotid or crural artery had been previously connected with the mercurial manometer of the kymographion. Immediately after each injection the mercury rose from 40 to 60 mm. above the point of average arterial pressure, and this rise was preceded by a slight fall. That this increase of pressure was due, not to an active absorption of fluid, but solely to the influence of cold, is proved on the one hand by its suddenness, and on the other by its non-appearance when the same quantity of warm water was injected. It was also shown by the curves that were taken of the arterial pressure, that, since neither the frequency nor strength of the pulse was changed, the effect was not a result of the heart's action. Hermann therefore considers the increased blood-pressure as a direct result of the introduction of a large amount of cold liquid into the stomach, inasmuch as the cold produces through the thin walls of the stomach an impression not only upon the minute arteries of that organ, but also upon the numerous vessels of those viscera which lie in its immediate neighborhood—the liver, spleen, diaphragm, omentum—and especially upon those of the loops of intestine, which are constantly in contact with it, whereby the arteries of these organs contract and cause a sudden rise in the arterial pressure.

In the above-mentioned case the existing kyphosis undoubtedly favored the production of the œdema.

A similar effect, that is, *contraction of the arteries, with an increase of the blood-pressure in the internal organs*, is produced when the peripheric temperature of the body is suddenly very much lowered by means of cold baths. That in this case the hyperæmia occurs more prominently and strikingly in the lungs than in the other organs, seems to be owing to the small power of resistance possessed by the exposed capillaries of the lungs. Another example is seen in the *collateral fluxion* in the lungs which occurs in the course of those diseases by which the circulation in more or less of the parenchyma of the lungs is impeded, giving rise to an increased blood-pressure in the remaining normal lung tissue. It may also be caused by atelectasis, compression and emphysema of the lungs, embolism of the larger arterial branches, and thrombosis of the pulmonary veins, likewise by distention of the abdomen, by accumulation of gas, fæces or fluids, or by tumors of the pelvic and abdominal cavities.

Pulmonary congestion is also caused by *irritants acting directly upon the respiratory organs*, for example, by *severe cold*, especially sharp winds, less often by a high degree of *heat*, but more frequently by the inhalation of *irritating* and *irrespirable* gases, such as chlorine, ammonia, and carbonic oxide. In these cases the hyperæmia is ascribed by some to a weakening and relaxation of the tissue surrounding the capillaries, and to the diminished power of resistance to the blood-pressure resulting therefrom (Niemeyer); others suppose a direct influence upon the arteries and capillaries, with relaxation of their walls; while others again (Seitz) believe that the irritation of the sensory nerves exerts a reflex effect upon the nerves of the capillaries.

Congestion of the lungs also follows *rarefaction of the air in the pulmonary alveoli*, consequent on narrowing of the glottis, as seen in laryngeal croup, in stenosis of the larynx caused by tumors, or in paralysis of those muscles which separate the vocal cords (the posterior crico-arytenoid muscles). It also accompanies *acute inflammatory conditions of the parenchyma*

of the lungs, croupous and catarrhal pneumonias, new-growths—such as cancer and sarcoma—acute and chronic tuberculosis, etc. In the latter conditions the hyperæmia seems to be due partly to a weakened state of the parenchyma of the lungs, with relaxation of the walls of the capillaries, and partly to the collateral fluxion.

Passive or obstructive hyperæmia—damming of the blood—takes place whenever the flow from the pulmonary veins is interfered with, and engorgement of the latter and their capillaries is produced. This condition is most frequently a result of *stenosis* and *insufficiency of the mitral valves*, whereby, on the one hand, the flow of blood from the left auricle is impeded, and, on the other, the latter is overdistended by the regurgitated blood, and on account of its thin muscular walls can but imperfectly resist the increased pressure. The flow through the pulmonary veins is inevitably checked thereby. Less frequently, and then only with approaching dissolution and commencing degeneration of the walls of the heart, does passive hyperæmia accompany disease of the *aortic* valves. Very rarely it is a result of compression of the pulmonary veins by tumors.

In like manner obstruction of the current, together with so-called passive œdema, occurs when *the propulsive powers of the heart encounter increased resistance*, as is the case in croupous pneumonia, or when a large pleuritic effusion has taken place rapidly.

Weakened action of the heart is a not less frequent cause, in consequence of which the blood is driven with diminished force into the arteries of the general circulation. Engorgement of the left auricle necessarily results, together with incomplete discharge of the blood from the pulmonary veins.

Weakened heart-action may be due to an *asthenic fever* occurring in the course of the acute exanthemata, typhoid or puerperal fever, etc. It may also result from less serious diseases occurring in old persons or in individuals who are weak and poorly nourished, as is the case with delicate children, in hard drinkers, and in the course of long-continued chronic diseases, or may be after a loss of the fluids of the body. Furthermore, in consequence of *fatty*, or of the much rarer *amyloid*, degeneration

of the heart-muscle, or it may follow inflammation of the heart—*myocarditis*. Again, there is stagnation of blood in the lungs during the *last hours of life*, whenever the death-struggle is long and the force of the heart fails gradually. In this case the blood is driven into the aorta and its divisions but slowly and incompletely, as is clearly shown by the increasing weakness of the pulse, the lowering of the temperature, and the cyanosis of the body.

Pathological Anatomy.

Acute congestion of the lungs is very often a temporary condition, and, therefore, not always the subject of an anatomical investigation. Yet if the congestion is very intense and widespread, death may result very rapidly—*apoplexia pulmonum vascularis*. The lungs are then found very much engorged, and crepitate less under the finger than the mere inspection of them would lead one to suppose. Their color is dark red, and the vessels distended; if a cut is made blood flows freely from the divided surfaces; the tissue itself is moist, succulent, relaxed, and its elasticity diminished. The mucous membrane of the bronchi is injected and covered with a gray or reddish frothy mucus.

In *chronic*, chiefly obstructive hyperæmia, the pathological changes are at first located principally in the lower lobes, and may thence spread gradually to the other portions. The portion affected is firmer and denser, and its weight is increased. Its color varies from a dark blue to a blackish red; dark red blood flows freely from its cut surfaces. The interstitial tissue is swollen, infiltrated with serum. The capillaries of the air-cells are dilated and tortuous; numerous extravasations of blood from the capillaries are frequently found in the parenchyma. These conditions are most marked in the dependent (posterior and inferior) portions of the lungs. The gradual narrowing of the alveoli, caused by the swelling of their walls, the dilatation and tortuosity of the vessels, to which more or less collapse of the lung, in consequence of incomplete respiration, is often added, cause the parenchyma of the lung to bear a certain resemblance, on section, to the tissue of the spleen. Hence the

term *splenization* of the lung, by which this condition is described.

Hyperæmia of the dependent portions of the lungs is most frequently met with in persons who are old and debilitated, or who have been weakened by serious attacks of illness (generally those accompanied by fever), and who have been long confined to bed, in whom the strength of the heart has been lessened and disturbances of the pulmonary circulation have occurred. It takes place in the most dependent portions, because there the circulation is the most sluggish, and is called *hypostatic hyperæmia*, or *hypostasis of the lungs*. This condition is generally bilateral, but it may be limited to one side, when, from some cause, the patient is obliged to lie constantly on the same side. There is quite often associated with it a cellular desquamative and fibrinous infiltration of the air-cells, generally of a lobar character, which gives to the cut surface a granular and drier and somewhat lighter-colored appearance, when compared with the rest of the parenchyma, which is moist and gorged with blood. This so-called *hypostatic pneumonia* is due to perverted nutrition of the parenchyma of the lung, the consequence of long-continued obstructive hyperæmia.

Bronchitis is often simultaneously present, for a hyperæmic and swollen condition of the mucous membrane of the bronchi, with increased secretion, is a necessary consequence not only of congestion, but also particularly of passive hyperæmia of the lungs. The reason for this is very apparent, when it is borne in mind that, according to Adriani, Heale, Reinay, and others, the bronchial mucous membrane is for the most part supplied with blood from the pulmonary artery, that without doubt the terminal capillaries of the pulmonary and bronchial arteries anastomose, and that the main portion of the blood from the bronchi is discharged from the lungs through the pulmonary veins.

A further result of active and passive hyperæmia of the lungs, and one due to the increased blood-pressure upon the walls of the capillaries, is the infiltration (Laënnec), transudation, or inundation (Rokitansky) of a serous fluid into the interstitial tissue and into the air-cells, whereby the capacity of the latter for air is seriously diminished. This condition, which we

call *pulmonary œdema*, may, though more rarely, appear *suddenly* during severe congestion of the lungs, and cause death in a short time. It is developed less rapidly during passive hyperæmia, and, so far as it is a consequence of hypostasis, represents *hypostatic œdema*. Indeed, in every patient suffering from passive congestion of the lungs there is more or less œdema, and even in the various diseases in which dissolution occurs slowly and the propulsive force of the heart gradually fails, pulmonary œdema supervenes during the last moments and very materially hastens death. It is a matter of some difficulty after death to distinguish between this latter œdema and that which has occurred during life as a result of congestion and engorgement.

Œdema of the lungs may also be the result of a *dropsical crisis*, when the serum of the blood containing less albumen in solution presses through the capillary walls into the interstitial pulmonary tissue and into the air-cells under slight intravascular pressure. This condition is associated with œdema of the subcutaneous tissue and with accumulations of fluid in the serous cavities after various hemorrhages or loss of the fluids of the body. Occasionally a so-called dropsical crisis and an active or passive hyperæmia unite in causing pulmonary œdema, the amount of which is necessarily increased thereby.

Pulmonary œdema occurring during a condition of general dropsy, runs, as a rule, a chronic course and rarely develops quickly throughout both lungs. That there is ever an idiopathic œdema of the lungs, as Laënnec asserts, which under certain conditions may last for months, is more than doubtful.

Œdema of the lungs develops either in a circumscribed portion of the organ, corresponding to that which is the seat of hyperæmia, thence spreading gradually over the rest or remaining stationary, or, less frequently, it involves from the beginning every portion of the lungs. An œdema which is due to passive hyperæmia and a dropsical crasis generally begins in the dependent inferior and posterior portions of the lungs and spreads upwards and forwards. When a patient has been compelled to lie upon one side the œdema is often confined to the corresponding lung, or is more marked in it than in the other. When the œdema is only an infiltration of the alveolar walls, with no effu-

sion into the alveoli, it may be termed *interstitial*—a condition which is more presumable from certain symptoms during life than it is demonstrable post-mortem, since before death has taken place more or less alveolar œdema is sure to have become associated with it.

An œdematous lung is bloated, and firmer and heavier than normal, does not collapse as much as a healthy lung when the chest is opened, and when touched conveys, according to Hasse, the sensation of a sponge filled with water. It is less buoyant than the normal lung, and in some cases sinks in water. In *acute* œdema the elasticity is retained, or only slightly diminished; pressure with the finger produces some crepitation, and the imprint remains for some time. In *chronic* œdema the elasticity disappears more and more, the tissue becomes softer and more friable, and the mark left by the finger lasts much longer.

The color varies according to the degree of the œdema and to the still present hyperæmia, whether acute or chronic. The more the œdema predominates, the paler is the lung, and its color a more bluish, red, or yellow-gray, while if the hyperæmia is the more marked feature of the two, the color is redder. In general dropsy the lungs may be very pale and entirely void of blood.

If a lung, the seat of acute œdema, be cut into, a very frothy, pale red, yellow, or colorless fluid, mingled with much air, pours out, and any blood which is mixed with it comes from the severed vessels. If there is much œdema, or if it has been of long standing, the discharge of fluid is more copious, but less mixed with air, and contains only very minute air-bubbles instead of the large ones which are found in acute œdema; or the parenchyma of the lung may be entirely void of air because the transuded serum has gradually driven it all from the alveoli and the smaller bronchi. If the fluid does contain a few large air-bubbles, they come from the larger bronchi. The parenchyma therefore crepitates but little or not at all, and is denser and turgid. As the blood escapes from the vessels the color becomes paler, and at last a dirty gray, which feature is especially striking when the œdema is a consequence of a serous dyscrasia. The serum which flows from the cut surface of such a lung is

also colorless, and should it have a yellow, or brown, or black tint, the latter is due to the admixture of biliary matter, or of brown or black diffused granular lung-pigment.

The bronchi contain a quantity of frothy liquid, and if the œdema has been acute the mucous membrane is injected, but if chronic, it is pale, except when catarrh coexists.

Besides the pulmonary hemorrhages which sometimes occur in the course of congestion of the lungs, and will be more fully described in the next chapter, we must mention still another condition which is observed as a consequence of passive hyperæmia, secondary to serious organic disease of the mitral valve, and is generally designated as *brown induration* (Virchow). In this case when the thorax is opened the lungs project prominently and collapse but little. They are very voluminous, firm, heavy, and inelastic, and crepitate but slightly under the finger. The surface of the lung varies in color from a yellow to a reddish-brown; a cut surface is pretty smooth, very hyperæmic, and discharges, if pressed, a yellow or brown liquid. The tissue is firmer than normal, its general color more yellow, orange, or rust-like, with red, brown, and black spots of variable size interspersed. The walls of the alveoli are thickened, the cavities of the latter are lessened, and contain less air; at individual points the parenchyma is void of air, of a uniform dark-brown color, and sinks in water. Together with these changes in the lungs, hemorrhagic infarctions are often present.

Rokitansky affirms that a considerable augmentation of the interlobular connective tissue is associated with these changes in the lungs, and both he and Skoda describe it as *hypertrophy of the lungs*. Andral thinks the coloring of the lungs is incidental to the abnormal induration caused by irritation of the tissue, an opinion in which he is partially sustained by Hasse (Hasse's *brown induration*). Dittrich and Heschl also recognize as the real cause of the pulmonary condensation an augmentation or hypertrophy of the connective tissue in the alveolar walls, to which Bamberger adds an abundant deposit of pigment. Virchow, who was the first to understand the relation of this affection to cardiac disease, declared, even earlier, the hypertrophy of the alveolar walls to be hypothetical, and ascribed the parenchymatous induration to an accumulation of granular or crystallized pigment in the interstitial tissue and in the epithelial cells, whereby the latter become of a uniform yellow color, or are filled with yellow, yellowish-red, brown, or blackish granules. Hence he designated this condition as *brown* or *pigment induration*. On the other

hand, Isambert and Robin, who call this affection *congestive carnification*, believe in a thickening of the alveolar walls produced by the deposition between the normal pulmonary tissue-elements of an amorphous finely granular substance containing numerous granules of hæmatine. Zenker denies the interstitial augmentation, especially the thickening of the membrana propria, seen by so many, which generally does not exist, and explains the induration as due to an extensive deposition of granular pigment, and to an enormous new formation of epithelium, as was also shown by Friedreich in one case. The collapse of the pulmonary alveoli is mechanically prevented by means of this accumulation of epithelial cells, which, according to Zenker, who is a disbeliever in alveolar epithelium, are drawn into the alveoli from the small bronchi during the deep inspirations of patients suffering from dyspnœa (Zenker's *pulmonary condensation* or *catarrhal condensation*). Buhl, and later also Virchow and Colberg, discovered, by microscopical examination, that the capillaries in the walls of the pulmonary air-cells presented considerable dilatations, of which some were simple and others varicose, with prolongations and loops, and knob-shaped processes, which projected into the alveoli, materially diminishing the capacity of the latter, and rendering their collapse impossible. According to these observers, vessels of this kind, when they are empty of blood, resemble thickening of interstitial tissue, and have given rise to the above-mentioned suppositions of pulmonary hypertrophy.

Judging from our own experience this condition is one of simple passive hyperæmia of the pulmonary capillaries, producing dilatation and elongation of the latter, with escape of blood-corpuscles (by diapedesis) into the alveoli, and into the interstitial tissue, with subsequent pigment change. Occasionally we have also found a slight and immaterial thickening of the interlobular connective-tissue, and of the muscular elements, just as it frequently occurs in chronic passive congestion of other organs, the kidney, liver, etc. In the same way we find here and there, but only as a complication, a new formation of cells in the alveolar epithelium, a sort of desquamation. These and similar appearances are sufficient to account for the different views of observers; they only occur as secondary changes or complications, and therefore of all the appellations received by them we prefer that given them by Buhl, *capillary ectasis*.

As already stated, this condition is most frequently associated with affections of the mitral valve, chiefly stenosis, and also, according to Zenker, when this is combined with insufficiency of the same valves, and with anomalies of the aortic valves. Moreover, this pulmonary lesion was met with by Rokitansky in a case in which, owing to a constricting induration, there was contraction of the divisions of the

branch of the pulmonary artery at its entrance into the upper lobe of the right lung. A similar case is reported by Colberg, in which there was considerable development of indurated connective tissue at the roots of both lungs, about and around the large vessels and bronchi, and extending quite a distance along the course of the latter. Isambert and Robin relate a case in which one lung was the seat of tuberculosis, while portions of the other presented indurations similar to those accompanying cardiac disease. Dittrich[1] also found this condition in one case unassociated with heart disease or any other lesion of the body, except a well-marked catarrh of all the air-passages. It occurred in a young woman, eighteen years of age, who was seized with a relapse of acute pulmonary catarrh, and died suddenly in an asthmatic attack.

Symptomatology.

A slight degree of active pulmonary hyperæmia produces no symptoms, for the reason, as Niemeyer very justly asserts, that the dilated capillaries offer a greater respiratory surface to the air, and the interchange of gases is facilitated and accelerated. The entrance of air is obstructed and the interchange of gases interfered with, first, when the hyperæmia is so intense that a transudation of serum takes place into the alveolar walls, and in a small measure into the alveolar cavities, and when the hyperæmia causes swelling of the mucous membrane of the smaller bronchi, with consequent diminution of their calibre. Such a condition unquestionably exists in persons with erethism of the heart after severe bodily exertion, moderate use of spirituous liquors, exposure to extremes of heat and cold, etc. These patients complain of a sense of fulness and oppression, of "bursting of the chest," and of the feeling that they cannot take a deep enough breath; flushing of the face, increased frequency of respiration, more marked pulsation of the carotids, and a dry short cough are the other symptoms of this condition, which, on account of the accompanying serous infiltration of the tissues, may be called interstitial œdema. *Physical examination* of the patient reveals nothing special beyond roughened vesicular respiration and a few moist râles. We would here state that we cannot but believe that when Lebert[2] repeatedly found a slight dulness over the posterior portions of the lungs in the

[1] Prager Vierteljahrschrift, 1851, p. 43, Anm.

[2] Klinik der Brustkr., Bd. I., p. 777.

"more interstitial œdema of scarlet fever and of idiopathic acute nephritis," he had to do with a complication having other anatomical changes.

The symptoms only become of an alarming nature when the hyperæmia has increased to such a degree that a transudation of fluid upon the free surfaces of the alveoli, that is, a true alveolar œdema, has taken place. Then the following phenomena present themselves: intense *dyspnœa*, with every now and then the appearance of suffocation; a sense of oppression, with terrible anxiety and fear of death; respiration almost too rapid to be counted; violent action of the heart, with strong pulsation of the carotids; congestion of the face, and a beating and throbbing in the temples. Furthermore, the patients are annoyed with *coughing*, which brings up *considerable quantities of a serous frothy liquid*, which is sometimes mixed with bright red blood, or tinted by the same a uniform red color.

Microscopical examination shows an abundance of well-formed corpuscles, which in the last-mentioned condition are uniformly diffused through the sputa, and are more probably the result of permeation through the uninjured but greatly distended capillary walls than of rupture of these latter. When rupture really does occur in consequence of severe paroxysms of coughing—after the inhalation of irritating gases, etc.—the blood appears more in the shape of streaks and spots.

On *percussion* the normal sonorous resonance partakes somewhat of a *tympanitic* character. *Auscultation* reveals during both inspiration and expiration no vesicular breathing, because it is concealed by the *abundant coarse and fine râles*, which are generally so loud as to be audible even at some distance from the patient. According to Oppolzer, when the alveoli become completely filled with fluid, instead of the just-mentioned râles and respiratory sounds, only a distinct buzzing and suction noise are audible. In such cases there may be some *dulness* on percussion, and, as remarked by Laënnec, a certain amount of *bronchophony*.

The more the alveoli become filled with fluid, the more incompletely does the interchange of gases go on, and the blood become surcharged with carbonic acid. The expression of the

patient is more anxious, his face is pale and livid, the lips blue, the body covered with a cold clammy sweat, the respiration panting, the jugular veins are swollen, and the pulse, which has hitherto been pretty full, becomes small, scarcely to be counted, irregular, and intermittent; finally, the patient becomes more quiet and sinks into a stupor. As a consequence of the paralysis of the bronchial muscles by the serous infiltration and the insensibility of the mucous membrane, owing to the gradual paralysis of the respiratory nerve-centres by the concomitant œdema of the brain, the patient is incapable of removing the contents of the bronchi, so that the coarse mucous râles in the trachea are audible at quite a distance.

In passive hyperæmia the sufferings of the patient, the dyspnœa and the distressing cough, are frequently of a far greater severity and duration, which is readily accounted for partly by their causative agents, especially the pre-existing cardiac affection, the incidental bronchial catarrh with swelling of the mucous membrane and obstruction of the bronchi with the secretions, partly by the subsequent degeneration of the heart, and also by the circulation having been retarded, instead of accelerated as in active hyperæmia. Therefore we frequently see, without any œdema being present, the dyspnœa and the terrible anxiety of the patient associated with swelling of the jugular veins, cyanosis of the face, cold and blue extremities, a small, quick, frequently irregular pulse, and furthermore, somnolence and dulness consequent upon a co-existing venous stasis in the brain.

If now œdema occurs, very naturally all these symptoms become intensified, especially if it comes on acutely, when death may speedily and suddenly result from suffocative effusion. On the other hand, the œdema may come on slowly and advance *pari passu* with the increase of the passive congestion and the hydræmic condition of the blood consequent upon the general nutritive disturbances. In such a case death takes place slowly, with the phenomena of imperfect respiration and carbonic acid intoxication.

In *pulmonary hypostasis* and *hypostatic œdema* the physical examination presents, on account of the decubitus of the patient, certain variations in the inferior and posterior portions of the

lung. These are either uniformly bilateral, or, if the patient has lain continuously upon one side, unilateral, or more pronounced upon one side than upon the other. According to the intensity of the hypostasis and œdema percussion yields a tympanitic sound or more or less distinct dulness, and auscultation weak vesicular breathing, dull, moist râles, or, if the condensation of the parenchyma be more considerable, sibilant râles, less frequently weak bronchial breathing. If the bronchi are obstructed and the respiration is superficial, the respiratory sounds in these portions of the lungs are either very indistinct or entirely absent.

In those cases in which the œdema of the lungs is the result of a watery condition of the blood, following nephritis and other similar diseases associated with abundant loss of fluid, it generally occurs slowly, and gives rise to symptoms which do not differ materially from those already depicted. An acute œdema may, however, though not frequently, take place in the course of an acute nephritis, as has been reported by Lebert.[1] Moreover, during a chronic nephritis, symptoms of an acute and well-marked paroxysmal character sometimes come on, which must be ascribed to œdema of the lungs, and I fully agree with Rosenstein[2] in believing the so-called *urœmic asthma* to be due to an œdematous swelling of the bronchial mucous membrane, and not to the effect of the blood-poisoning upon the nerves governing respiration. In fact, that condition is nothing more nor less than an *interstitial* œdema of the lungs, with infiltration and swelling of the mucous membrane of the terminal bronchi. I have hitherto only observed this œdema in those patients in whom during their illness I could, from the presence of symptoms which are described by Bartels[3] as characteristic of primary cirrhosis of the kidneys, diagnosticate that affection and subsequently verify it by post-mortem examination. It is certainly surprising that œdema of the lungs is met with just in those patients who, while having the contracted kidneys, do not suffer from extensive œdema of the surface, or from fluid accumula-

[1] Klinik d. Brustkrankheiten, Bd. I., p. 785.

[2] Pathol. u. Therapie der Nierenkrankheiten, 2. Aufl., p. 196.

[3] *Volkmann's* Sammlung klinischer Vorträge, Nr. 25.

tions in the large internal cavities of the body, until death is near at hand. Judging from my own experience, these cases of nephritis are always found to have associated with them some other complicating pulmonary affection, for instance, emphysema, bronchial catarrh, atelectasis, etc. Notwithstanding the destruction of many of the capillaries of the kidneys the secondary hypertrophy of the heart is capable for a long time of forcing a large amount of pale watery urine through the vascular tufts which still remain. When, however, the heart-action becomes weaker, there occurs, without any apparent special reason therefor, unless it be the incidental diminution of the secretion of urine, passive hyperæmia of the lungs, producing a serous effusion into the already affected pulmonary and bronchial tissues, and the well-known terrible paroxysms of so-called uræmic asthma. Even the eating of a hearty meal may, by distending the abdominal organs and thereby interfering with the movements of the diaphragm, give rise to passive congestion of the thoracic viscera, and if disease of the lungs is already present, to paroxysms similar to those just mentioned.

On November 21st, 1871, I was consulted by a gentleman, fifty years of age, from the country, who, according to his own opinion, had enjoyed perfect health up to six weeks previous. The only ailments he had suffered from were repeated attacks of bronchial catarrh during the winter seasons. On the 10th of October, he sought medical advice for the first time, on account of headaches recurring every night, and which, he said, were successfully treated by the administration of quinine. On the 19th of November he had, without any appreciable cause, a severe attack of dyspnœa, with very intense headache. Two days later I was consulted. The patient was a man of medium size, pretty corpulent, and having a well-marked panniculus adiposus, although he declared that during the last few months he had lost twenty-six pounds in weight. He had long been troubled with constipation. The stomach and intestines were much distended with gas. The face was congested, the præcordial dulness enlarged, the impulse of the heart against the thoracic parietes increased, and of a lifting character, very distinctly to be seen and felt in the seventh intercostal space, one finger's breadth outside the linea mammillaris. The heart sounds were clear, the second aortic sound was much intensified and accentuated, the pulse full and hard. Bronchial catarrh pretty well diffused over both lungs, some œdema at both ankles. Urine very pale, moderately albuminous, and without any sediment of epithelial or fibrinous casts; spec. grav. 1.012. Quantity for the twenty-four hours forty-four ounces. *Diagnosis: contracted kidneys, with secondary hypertrophy of the left heart.* As the attacks of dyspnœa

recurred daily, and the administration of quinine was of no avail, the patient returned on the 10th of December to Amsterdam, and placed himself under my care. I now remarked that the attacks came on daily towards seven o'clock in the evening, about one or one and a half hours after the ingestion of a pretty hearty meal. While they lasted, the act of inspiration was laborious and wheezing, and during its performance the auxillary muscles of respiration were called into action; expiration, also, was of an active nature, and aided by the abdominal muscles. Very abundant, moist, subcrepitant râles were audible, the action of the heart was irregular, and the pulse small and frequent. The attacks at first lasted several hours, but later, they were considerably shortened by the administration of an emetic (a grain of tartar emetic and twenty-three of ipecac in a single powder). Sinapisms also were applied to the chest, mustard foot-baths twice a day, and decoction of senega with thirty minims of anisated spirit of ammonia;[1] furthermore, the hearty meal, which had hitherto been taken at five o'clock, was divided into several smaller ones. Under this plan of treatment, and the use of infusion of digitalis during the day, with attention to the free action of the bowels, the attacks of dyspnœa ceased, and the patient departed at the end of twelve days for his home. I have heard nothing of him since, except that he died some months afterwards with symptoms of general dropsy.

Several months before, I met with a similar case in my clinic, in the person of a woman, forty-five years of age, having kyphosis to a marked degree, and who entered the hospital on account of her asthma. In this case also the diagnosis of contracted kidneys with secondary hypertrophy of the heart was readily made. The asthmatic attacks were, without exception, preceded by a diminution of the secretion of urine, which after the attacks resumed its normal volume, or even very considerably exceeded it, a circumstance which was also observed in the first case, although, not with the same precision, the patient being in private and not in hospital practice. In both patients there were no symptoms except the headache, in the first case, which could be called uræmic, and this experience has caused me to agree with Rosenstein in denying a blood-poisoning in the so-called asthma uræmicum. I could present several other cases of a similar kind, but an account of them here would be out of place.

Course and Termination.

Hyperæmia may, as already stated, set in *acutely*, and when only of moderate intensity disappear, to return again repeatedly under favoring circumstances. When the onset is sudden and severe, the gravest symptoms may come on in a short time, and, if the patient is not speedily relieved, death may ensue through suffocative effusion in a few hours. This acute hyperæmia, with

[1] Oil of anise, 1 part; alcohol, 24 parts; water of ammonia, 5 parts.—*German Ph.*

consequent œdema, is generally of a congestive character; less frequently it occurs in the course of passive hyperæmia, or as an accompaniment of general dropsy and contracted kidneys. Recovery from an acute hyperæmia is generally associated with a sero-frothy expectoration, which is wanting, as a rule, in interstitial œdema. In other cases it follows an increased secretion of urine. *Slowly developing* and *chronic* hyperæmia and œdema are generally the result of passive congestions, or are an expression of a watery condition of the blood. In these cases, under the influence of medical treatment, a gradual improvement will take place, only to be followed again by an aggravation of the disease; death comes slowly, though an acute fatal œdema may suddenly, and without any apparent cause, supervene upon a chronic hyperæmia.

Prognosis.

Acute hyperæmia of the lungs, due to erethism of the heart, or to the injurious effects of any of the above-mentioned causes upon the respiratory organs, or when associated with inflammatory changes and new-formations in the lungs, does not, in the majority of cases, entail any direct danger, as it speedily disappears either spontaneously or after proper dietetic and medical treatment. It not unfrequently gives rise to hemorrhages, which will be spoken of in the next chapter, and which, likewise, as a rule, are speedily recovered from. The prognosis of *passive hyperæmia* depends upon the disease of which it is a result.

Œdema of the lungs is always a critical symptom when it occurs as the last scene in certain diseases, or as an indication of obstructions in the circulation which cannot possibly be removed. When it comes on suddenly and severely in the course of an acute hyperæmia, a fatal termination results in the majority of cases; subacute œdema of a congestive character will yield to appropriate treatment. The acute interstitial œdema, which sometimes occurs with contracted kidneys, does not seem to entail a directly fatal result, for I have never yet heard of such a case.

Diagnosis.

The diagnosis of hyperæmia of the lungs is based principally upon the exciting causes, which will also determine whether that condition is of an active or passive nature. Furthermore, the above-mentioned symptoms must be carefully considered, and should they be indistinctly expressed, the disease can often only be presumed. The advent of alveolar œdema during pulmonary hyperæmia is determined partly by the degree of the dyspnœa, partly by the copious, thin, sero-frothy, reddish expectoration, and also by the results of percussion and auscultation, especially the widely diffused, moist, subcrepitant, and mucous râles. When the œdema results from a watery condition of the blood, the anasarca and the effusion of fluid into the serous cavities are to be considered in the diagnosis. In the œdema associated with contracted kidneys, the condition of the urine is of special importance: the amount of albumen, the low specific gravity, the light color, the decrease of the urinary secretion before, and its increase after the attack. The demonstration of some form of hypertrophy of the heart, or of pre-existing pulmonary and bronchial affections, is also of great moment.

The diagnosis of *capillary ectasis* can only be made with certainty in very few cases, because the bloody expectoration and dyspnœa may depend upon passive hyperæmia, due to co-existing heart disease, although it is true that these symptoms are more intense in capillary ectasis, owing to the alveoli being much diminished in capacity, and hence capable of admitting less air. This is, however, only a quantitative difference, which cannot be of much diagnostic value. Careful attention should be directed to the existing mitral disease, and also, according to Bamberger, to the very marked and uniform diminution of resonance over the whole thorax, while vesicular respiration still continues to be audible, though accompanied by the characteristics of a catarrh; and further, to the mucous, and frequently bloody expectoration, which often contains considerable quantities of pigment cells. According to Zenker, in the gravest cases dulness on percussion is materially increased, and the respiratory sounds are of a decidedly bronchial character, so

that the slow progress of the disease is the only feature which distinguishes it from a croupous pneumonia. A differential diagnosis between capillary ectasis and pulmonary infarctions, under these circumstances, is scarcely possible.

TREATMENT.

The exciting causes should if possible be removed; thus if a predisposition to increased action of the heart exist, all mental excitements, bodily exertions, hot and exciting drinks, as well as the respiration of a cold, hot, dusty, or polluted atmosphere should be most carefully avoided. In those cases in which the slightest causes produce erethism of the heart, I recommend, together with a good nourishing, non-exciting diet, the use of the milk, whey, and grape cures, and that the patient should remain as much as possible in the fresh air and wash his breast every morning with a cold wet sponge, followed by energetic rubbing with a rough bath-towel, bath-glove, or brush. Acid draughts should be given: cream of tartar, Haller's acid elixir,[1] sulphuric, muriatic, or phosphoric acids, and under certain conditions tincture of digitalis, fifteen drops four times a day, alone or combined with Haller's acid, or twenty drops four times a day of the vinegar of digitalis.[2] In the prophylaxis the aforementioned causative agents should be carefully borne in mind. Where passive hyperæmia occurs in consequence of severe asthenic fever, the indications are, a frequent change of the patient's position and the administration of excitants and stimulants, such as the preparations of ammonia, camphor, musk, and strong wines. In *very grave acute hyperæmia of the lungs*, with acute œdema, the following important therapeutical indications are to be met. As *diminution of the volume of the blood* relieves the blood pressure and repletion of the vascular system, the further transudation of serum may thereby be prevented, and the absorption of the exuded fluid in the lungs promoted.

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[1] Pure sulphuric acid, 1 part: add by drops, while stirring, to alcohol, 3 parts.—*German Ph.*

[2] Digitalis leaves, cut, 1 part; alcohol 1 part; pure vinegar, 9 parts. Macerate for eight days, express and filter.—*German Ph.*

Hence, in a moderately well-nourished patient a bold venesection should be practised. A condition of general dropsy, during chronic nephritis for example, contraindicates this procedure, as then the blood is already in an extremely watery condition, which bloodletting would only serve to increase. It is different, however, in interstitial œdema associated with chronic contracted kidneys. In this case the patient is generally in fair health and in good condition, and the blood less hydræmic, as the loss of albumen with the urine is evidently less than with the enlarged kidneys, which are much more prone to give rise to a condition of general dropsy. Therefore, if emetics and derivations to the skin and bowels, etc., prove of no avail, and the œdema is of so severe a grade as to endanger life, there should be no hesitation in resorting to a general bloodletting.

In those cases also in which symptoms of incipient œdema of the brain set in, venesection is indicated, whereby the same advantages are obtained for the brain as for the lungs, that is, a resorption of the effused fluid. General bloodletting is contraindicated only in those cases in which, according to Oppolzer, somnolence is associated with an irregular pulse and an occasional intermission of respiration, indicating an œdema of the medulla oblongata, for the irregularity of both pulse and respiration would thereby be increased, and death might occur during the operation. This procedure is also indicated in cases of extensive acute croupous pneumonia, when the healthy portions of the lungs become intensely hyperæmic and the heart has not sufficient force to propel the blood through them. In such cases a diminution of the volume of blood will be a direct advantage, especially if in debilitated individuals the venesection be preceded and followed by the free administration of stimulants. I have employed this mode of treatment even in old, not particularly robust patients of about the age of fifty years, who had already been given up by me, and who were nevertheless saved by this means.

A second indication is the *removal of the fluid exuded into the alveoli* by the *promotion of expectoration.* This is best fulfilled by emetics, such as tartar emetic combined with ipecac, sulphate of copper and hydrochlorate of apomorphia. The latter

is better employed subcutaneously, from one-eleventh to one-fifth of a grain at a dose, and should have the preference in very urgent cases on account of its prompt and certain action, and because of the absence of all unpleasant accessory effects. Emetics are, however, contraindicated whenever the patient's strength is at a minimum, the nervous force diminished, and the near approach of death threatens, as they would then only hasten the collapse. When the hyperæmia comes on less acutely, and the fluid exuded into the alveoli and bronchi is only partially expectorated because the contractility of the bronchi and the sensibility of the mucous membrane are diminished, then it is well to use ipecac, or such expectorants as stimulate and excite the mucous membrane, such as arnica, seneka, benzoic acid, etc.

Derivation to the skin, intestines, and, under certain circumstances, to the *kidneys*, may be employed as an adjunct to the other modes of treatment in acute œdema. Likewise in the gravest cases of active hyperæmia and acute œdema, besides venesection it is well to employ mustard plasters on the breast and inferior extremities, warm hand- and, if practicable, foot-baths of lye or mustard, and embrocations with spirit of mustard.[1] The derivant mode of treatment is also of value in those cases in which, venesection having been practised, hyperæmia with œdema again sets in, or in which for the above-mentioned reasons bloodletting is contraindicated. In strong individuals derivation may be practised upon the intestines by means of calomel and jalap, senna, castor or croton oil, etc., or by stimulating injections into the rectum.

Derivation to the kidneys is chiefly indicated in acute œdema with cardiac disease, and the most efficient means for that purpose is infusion of digitalis in doses of one or two teaspoonfuls[2] every two hours, the diuretic effect of which is entirely owing to the increased arterial pressure produced by it. Lebert recommends digitalis in affections of the heart associated with kidney disease. I myself gave it with excellent results in the two cases already cited, and also in a third, especially when the pulse was

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[1] Oil of mustard, 1 part; alcohol, 50 parts.—*German Ph.*

[2] Double these doses for the infusion of the British Pharmacopœia.

increased in frequency and irregular. In cases of contracted kidneys, where the œdema of the lungs depends likewise upon a passive congestion of those organs which the heart strives to remove by force, the administration of digitalis, by decreasing the frequency of the heart's action, controlling its irregularity and increasing its energy, will undoubtedly be followed by good results. Digitalis is contraindicated whenever there are disturbances of innervation on the part of the medulla oblongata, especially irregular respiration, as it can then only increase these threatening features. Under such circumstances it would, according to Lebert, be justifiable to administer digitalis combined with stimulants (the preparations of ammonia, camphor, benzoin, etc.), or, still better, to give the latter for a while until the innervation had been revived and restored, and then follow with the digitalis. All other diurectic means are of no avail in acute œdema, even in cases of contracted kidneys, in which the attacks are preceded by a diminished urinary secretion, since the latter is only due to a diminution in the force of the heart's action.

A final indication is furnished by the *weakened innervation accompanied by irregularities in the respiratory acts.* The most effective means under such circumstances, besides expectorants, are the preparations of ammonia, ether, camphor, musk—the latter also given subcutaneously, as the tincture of musk,[1] in doses of from twelve to fifteen drops—and the strong wines—Madeira, port, etc.

As a special remedy for disturbance of the circulation and respiration, Oppolzer recommends quinine, and Traube speaks highly of the acetate of lead—three-quarters of a grain every hour—for œdema of the lungs.

A limitation of the above-mentioned therapeutical indications is necessary in *chronic passive hyperæmia* with œdema. Thus, venesection is applicable only rarely, and in the most extreme cases, because the hydræmia is increased by it, and the disposition to serous exudations thereby augmented. Bloodletting

[1] Musk, 1 part: triturate thoroughly with distilled water, 25 parts; add dilute alcohol, 25 parts; prepare by maceration.—*German Ph.*

is likewise injurious during a condition of general dropsy, as in Bright's disease. In the latter case I have seen good results follow from wrapping the lower extremities in warm, wet cloths, then covering these with blankets, and causing the patient to pass several hours daily in this condition. In cases of cardiac disease the treatment by derivations is worthy of trial, and in moderately strong persons by means of the bowels or kidneys. Digitalis is undoubtedly here also the best diuretic, as it regulates the action of the heart, increases the blood-pressure in the aortic system and thereby frequently gives rise to free diuresis. It is also occasionally possible to rid the patient of his general dropsy and pulmonary œdema, if only for a short period, by means of other diuretics (cream of tartar with nitre, acetate of potash, and boro-tartrate of potash). Expectorants also will be of service in the treatment. It is rational to treat the fundamental disease at least symptomatically, above all to counteract the watery character of the blood by means of tonics, quinine and iron, and an appropriate diet, as well as to excite the gradually failing forces of the heart by means of stimulants.

HEMORRHAGES OF THE LUNGS.

Haller, Opuscul. pathol., Obs. XVI. hist. 1.—*Latour*, Hist. philos. et méd. des hæmorrhagies, 1815, I., 240.—*Cruveilhier*, Essai sur l'anat. pathol., 1816, II., p. 103, and Anat. pathol., 1828, III., and Dict. en XV., Vol. III., 278.—*Hohnbaum*, Ueber den Lungenschlagfluss, 1818.—*Laënnec*, Traité de l'auscultation méd., 1819.—*Bouillaud*, Archiv. géner., 1826, T. 12, p. 392.—*Townsend*, Cyclop. of Pract. Med., 1833, I., 134.—*Brecheteau*, Archiv gén., 1836, 2 Ser., T. 12, p. 400.—*J. Hope*, Principles and Illustrations of Morbid Anatomy, London, 1834. —*Chomel* et *Reynaud*, Dict., 1837, en XXX., Vol. XV., 125.—*Gendrin*, Traité philos., 1838, T. I., pp. 136 and 637.—*Andral*, Anat. path., II., p. 139.—*Hodgkin*, Lect. on morb. anat. II., p. 118.—*Hasse*, Patholog. Anatomie, 1841, I., p. 316.—*Rokitansky*, Lehrbuch der path. Anat., 2. Abdr., 1842, III., p. 72, 3. Aufl., 1861, III., p. 76.—*Graves*, Clin. med., 1843, 22.—*Williams*, Med. Times, 1843, Oct. 28.—*Bochdalek*, Prager Vierteljahrschr., 1846, Bd. X., I., 2.—*Wunderlich*, Handbuch der Pathologie und Therapie. Stuttgart, 1846, Bd. III.—*Heusinger*, Casper's Wochenschrift, 1847, Nrs. 3 and 4.—*Dittrich*, Beiträge zur patholog. Anatomie der Lungenkrankheiten. Erlangen, 1850.—*Stanhope-Templeman-Speer*, Monthly Journ., 1851, Oct. (Schmidt's Jahrb., Bd. 73, p. 176).—*Hoffmann*, Ueber die Blutung in den Luftwegen, Diss. Erlangen, 1852.—*Virchow*, Gesammelte Abhandlungen, 1856, p. 221.—*Heschl*, Prag. Vierteljahrschr., 1857, Bd. II., p. 21.—*Scoda*, Allgem. Wien medic. Zeitung, 1860, No. 8; Prager Vierteljahrschr., 1861, Bd. I.; Wien med. Zeitung, 1862, Nr. 33.—*Panum*, Virchow's Archiv, 1862, Bd. XXV., pp. 308 u. 433.—*Gerhardt*, Würzb. med. Zeitschrift, 1864, Bd. V., 221.—*Bürger*, Verhältniss der Bronchial- und Lungenblutungen zur Lungenschwindsucht. Dissert. Tübingen, 1864.—*Hopf*, Zur Diagnose des hämorrh. Infarktes. Dissert. Tübingen, 1865.—*Ogston*, Brit. and For. Med.-Chir. Rev., 1866, Apr. (Hirsch u. Virchow's Jahresb. 1866, II., p. 91).—*Oppolzer's* Vorlesungen über spec. Pathologie und Ther., von Stoffela, 1866, I., p. 501.—*Ott*, Niemeyer's klinische Vorträge über Lungenschwindsucht. Berlin, 1867.—*Rindfleisch*, Lehrb. der pathol. Gewebelehre. Leipzig, 1867 und 1873.—*Traube*, Berliner klin. Wochenschr., 1867, Nr. 46.—*Oppolzer*, Ueber Haemoptoë. Wien med. Presse, 1868, p. 42 et seq.—*Rasmussen*, Hospitals Tidende, 11 Jahrg., p. 33 (Virchow und Hirsch, Jahresb., 1868, II., 90).—*Seligmann*, Ueber den hämorrh. Infarkt. Diss. Berlin, 1868.—*Niemeyer*,

Berl. klin. Wochenschr., 1869, Nr. 17.—*Burdon-Sanderson*, The Lancet, 1869, I., p. 523.—*Scoda*, Wien med. Presse, 1870, p. 13—*H. Weber*, Clin. Society Transactions, 1870 (Med. Centralbl., 1870, p. 93).—*Peter*, L'union méd., 1870, 36.—*Johnson*, Brit. Med. Journ., 1870, Feb. 12.—*Bradburg*, Ibidem, 1871, Sept. 2.—*F. von Niemeyer*, Specielle Pathologie und Therapie, in den verschiedenen Auflagen und 9. Aufl. herausgegeben von Seitz, 1874.—*Perl* und *Lipmann*, Virchow's Archiv, Bd. 51, p. 552.—*Sommerbrodt*, Untersuchungen über Lungenblutung. Centralblatt f. med. Wissensch., 1871, Nr. 43, und Virchow's Archiv, Bd. 55, p. 165.—*Rasmussen*, Hospit. Tidende, 1871, und med. Centralbl., 1871, p. 715.—*Cohnheim*, Untersuchungen über den embolischen Process. Berlin, 1872.—*Lebert*, Klinik der Brustkrankheiten, 1874.—*Penzoldt*, Deutsches Archiv, Bd. XII., p. 13.

Hemorrhages from the respiratory organs—*Hæmoptoë*—and expectoration of blood—*Hæmoptysis*—are only symptoms of various morbid processes, and arise partly by rupture of vessels, partly by diapedesis taking place in the larynx, trachea, the large and small bronchi, or in the parenchyma itself of the lungs. *Bronchial hemorrhages*, especially those occurring in the smaller and terminal bronchi, are by far the most frequent, whilst hemorrhages of the larynx, trachea, and larger bronchi are comparatively rare; less frequent, too, than the bronchial, is parenchymatous hemorrhage of the lung. In this case the hemorrhage is either confined and sharply outlined, causing displacement, but no destruction of the parenchyma—*hemorrhagic infarction*—or it is abundant and more diffuse, causing destruction of the lung tissue, with the formation of cavities—*pulmonary apoplexy*.

Disregarding for the present hemorrhages of the trachea and larynx, we will confine our discussion to those of the bronchi and lungs.

HISTORY.

In early times, when disease was symptomatically regarded, only those hemorrhages were recognized which were characterized by the expectoration of blood, and, according to the amount of the latter, two morbid processes were distinguished, *hæmoptysis* and *pneumorrhagia*. Pulmonary hemorrhages were first classified anatomically by Laënnec, who divided bronchial hemorrhages (exudation, diapedesis of blood upon the free surface

of the bronchi) from other pulmonary hemorrhages, the most serious of which represent *pulmonary apoplexy*, and the less serious *hæmoptoic engorgement* (infarction). In his description of the anatomical characteristics he dwells particularly upon the differences between the latter and pneumonic infiltrations, and post-mortem engorgement; he describes the symptoms fully, and establishes a sure diagnosis. It has, in fact, been impossible since then to make any material addition to Laënnec's excellent anatomical description; yet concerning the mode in which the hæmoptoic infarction is produced, a subject passed over in silence by Laënnec, the greatest variety of opinions have been advanced, and only the investigations of the last few years have satisfactorily explained this process. Bochdalek believes that the infarction depends upon an inflammation of the smaller and terminal branches of the pulmonary arteries in the diseased area, and that the plugs of fibrine found by him in the calibre of the vessels represent the inflammatory products thrown out from the lining membrane of the arteries. Engel declares it to be a morbid process resulting from inflammation or from inflammatory stasis. Dittrich attributes infarction to a morbid condition of the pulmonary arteries, consisting either in a very great dilatation of the calibre, or in a fatty degeneration of their walls, extending even to the terminal branches, or in a sort of subacute inflammation with fibrinous exudation between the coats of the vessels, and consequent thickening (arteriitis chronica). These changes which, especially when mitral insufficiency and stenosis are present, are associated with subsequent dilatation and hypertrophy of the right ventricle, are looked upon by him as a result of an increased blood pressure in the pulmonary artery, whereby the resisting power of the walls of the vessels is diminished. Bouillaud, Payet, and Dittrich consider the fibrinous plugs found in the branches of the pulmonary artery as secondary products, and as a result of the blood-stasis and obstructed capillary circulation in that portion of the lung which is the seat of the infarction. Heschl looks upon hemorrhagic infarction as a circumscribed inflammation of the lung, generally accompanied by epithelial changes, and occasionally by hyperæmia and hemorrhage—the latter dependent partly

upon predisposition, and partly upon the obstruction of the pulmonary circulation caused by the heart disease. Virchow's investigations of thrombosis and embolism first led to new views concerning the development of infarction. Although Virchow did not succeed in producing artificially in animals a condition similar to hemorrhagic pulmonary infarction in man, yet he showed it was probable that the latter was due to arterial embolism. Rokitansky was the first to declare positively that all infarctions were caused by occlusion of the finest branches of the pulmonary artery and their capillaries, "inasmuch as a collateral hyperæmia was thereby produced, resulting in hemorrhage and exudation." Cohnheim has recently, by means of his experimental researches, confirmed the theory that infarctions arise from emboli, and has followed their development step by step with the microscope. The clinical diagnosis of infarction has been facilitated chiefly by Wunderlich, Niemeyer, Gerhardt, and others.

ETIOLOGY.

Small hemorrhages take place by diapedesis, when the blood-corpuscles pass through the uninjured capillary walls, large ones by rupture of the vessels.

Bronchial hemorrhages, the most frequent form met with, rarely have a *traumatic* origin. They are caused, for the most part, by all those agents which engender *hyperæmia* of the bronchial mucous membrane, whether that condition be due to *increased afflux of blood or to passive congestion.* Hence, they not infrequently come on during severe attacks of acute bronchial catarrh with violent coughing, also during whooping-cough, the acute exanthemata, acute pneumonia, etc.; they are also produced by irritation of the air-passages caused by inhalation of irritating gases, by the effects of extreme degrees of heat and cold, by severe strains and bodily exertion, and especially by over-exertion of the respiratory organs by shouting, speaking, and singing. They are caused, furthermore, by the deposit of tubercles in the bronchial mucous membrane and in the parenchyma of the lung, and also by serious disorders of the circulation resulting from heart disease.

Among the bronchial hemorrhages due to acute hyperæmia should also be classed certain *vicarious* ones; such, for instance, as are supposed to follow cessation of hemorrhoidal bleeding, or of the menses. The first may certainly be ignored, for they exist only in the imagination of certain physicians, and the latter should only be accepted with great caution. Laënnec indeed quotes from Tulpius (Lib. II., Cap. II.) that such a periodical expectoration of blood has been known to come on at regular intervals during a period of thirty, and even forty, years, and Seitz explains it as a hyperæmia caused by reflex irritation. Nevertheless whenever a bronchial hemorrhage occurs before, during, or immediately after menstruation, or in the absence of the latter, it should be considered sufficient to call for a careful examination of the chest, for the lungs and bronchi of such persons are seldom normal; great fragility of the walls of the vessels, tubercles, or the primary elements of phthisis are the cause in many cases. Of greater significance are certain changes in the lung tissue. These consist in *softness and looseness of the parenchyma*, in consequence of a chronic inflammatory condition, or in anomalies of the vessels, such as a *flaccid and swollen state of their walls* (certainly more presumptive than demonstrable), or in *cell-proliferation* in the outer coat of the vessels. To these changes, so very prejudicial to the strength of the vessels, must be added the ulcerative and sloughing processes associated with the cavities of phthisis and bronchiectasis, with gangrene, abscesses, cancer of the lung, etc. Hemorrhage may also take place in a cavity, without extension of the ulcerative process to the wall of a vessel, if one of its mural arteries becomes the seat of an aneurism, and subsequently, especially if its efferent branches are occluded, is ruptured by the increased intravascular pressure during an attack of coughing.

Furthermore, an altered condition of the blood, entailing impaired nutrition of the vascular parietes, must be considered of etiological moment, a condition which is manifested in scorbutus, hæmophilia, scarlatina, typhus, variola, etc., by bleedings from other portions of the body (petechiæ, ecchymoses, hemorrhages from the gums and nose).

A good deal of difficulty, however, not unfrequently attends

our investigations and endeavors to find plausible etiological forces for certain, often very sudden, bronchial hemorrhages. This is especially the case when otherwise healthy and robust individuals, in whom the most thorough examination had previously failed to demonstrate any disturbances of the respiratory organs, and whose family history for several generations gives no trace of phthisis, are suddenly, without any exertion or other directly demonstrable pernicious influence, seized with a hæmoptysis which is frequently very copious.

Those cases also of hæmoptysis give much difficulty which occur in young persons between the age of puberty and the middle of the twentieth year, who have feeble constitutions, pale transparent skins, circumscribed red patches on the cheeks, and conspicuous temporal veins. They have generally grown rapidly, are of slender form, weak development of muscular and osseous systems, with rachitic or narrow, long, flat chests. Such persons are frequently the children of phthisical, debilitated or syphilitic parents, and many of them have suffered during childhood from scrofula and frequent epistaxis. In those cases in which the patients have not previously had any affection of the respiratory organs, it is allowable to suppose a primary general disorder of nutrition, especially one, although not yet anatomically known, of the walls of the vessels. Very often, however, changes have already taken place in the lungs: catarrhs, peribronchial, desquamative, pneumonic processes or tubercles, which, with their frequently insignificant symptoms, are long overlooked by the patient and his relatives, and are first appreciated by the careful physician.

Sex exerts no influence. Hemorrhage may occur at any *age*, although it is most frequently met with in youthful and middle-aged people. In old persons and in children bronchial hemorrhage is less frequent. I have seen two cases of profuse bleeding, with caseous pneumonia, in children aged respectively three and four years, and Lebert reports a fatal hemorrhage from a cavity in a child two years old.

Hemorrhagic infarction presents a more or less extensive, generally cuneiform, sharply defined infiltration of blood situated in the hilus, or more often at the periphery, of the lung,

and, as was first pointed out by Laënnec, is most frequently met with in cases of heart disease. In fifty-nine cases of infarction Bochdalek found organic disease of the heart thirty-eight times. They are especially those affections of the heart which produce dilatation of its right side ; hence mitral lesions, less frequently myocarditis and degeneration of the muscle. Moreover, it also occurs with pulmonary emphysema, and according to Dittrich, with senile and early acquired atrophy of the lungs, and also with thrombosis of the peripheric veins of the body.

Since Virchow's experimental researches, in which particles of various substances, such as fibrine, muscular tissue, elder pith, etc., were introduced into the circulation of animals, it can no longer be doubted that pulmonary infarction is due to embolism, and that its origin must be sought for in that portion of the circulation which leads to the lungs, in the right heart, in the diploë of the skull, etc., or in the general venous circulation, excepting the portal system. In most cases, indeed, the original thrombosis can be found in the peripheric veins of the body, in the right heart, between the columnæ carneæ, at the apex of the ventricle, or in the auricle.

The coagula in the right heart in cases of organic disease of the mitral valves are almost always the result of a retarded and weakened action of the heart, that is, as soon as the compensation on the part of the right ventricle becomes insufficient. These coagula are but seldom the consequence of myocarditis, or of roughness, or any other organic change in the rarely diseased tricuspid valves.

Although the source of pulmonary infarction is now so well known, through the excellent observations of Virchow, yet the *modus* of its formation has long been a matter of controversy. Some consider it to be due to the increased pressure in the collateral branches caused by occlusion of a branch of the pulmonary artery. Niemeyer founds his explanation upon Ludwig's statement, that the tension within the artery below the point obstructed by the embolus is at first diminished, and that in consequence of the sluggishness of the stream thus produced, the blood-corpuscles accumulate in and occlude the capillaries, and that the latter acting then as blind appendices to the artery

cause the pressure within it to increase to such a degree that rupture takes place.

Cohnheim with his usual ingenuity has made this process the subject of experimental research, and has followed it in its different phases under the microscope.

He selected for the purpose of experiment the frog's tongue, which, as is well known, when drawn out and retained in position, presents a large field adapted to microscopical observation. As emboli, he made use of an emulsion of blackened wax-pellets, which was injected through a small wound which could be easily closed again, either in the apex of the heart or into the aorta. When obstruction, as actually seen under the microscope, takes place, the blood flows with greater rapidity through a collateral branch given off at a point anterior to the impediment, spreads through its capillaries, and returns through the usual veins without giving rise to any vascular rupture. In the occluded arterial branch, however, the blood becomes stagnant both in front of and behind the obstacle, and should it happen that beyond the plug, between it and the capillary area of the artery, a branch is given off which anastomoses with another artery, the capillary area of the obstructed one may, without suffering any disturbance, be supplied through it with the necessary quantity of blood, and no infarction result. Should, however, such a communicating arterial branch be wanting, and the occluded artery divide after a longer or shorter course into its ultimate capillaries—that is, should it be what Cohnheim calls a "*terminal artery*"—complete stagnation takes place in the portion of the artery behind the embolus, in the capillaries, and in the veins as far as the point at which they are joined by other veins which have been supplied by another artery. In a short time, however, the blood flows backwards from the veins of the free arteries into the veins of the occluded one, and thence into its capillaries and into the artery itself, until all these vessels are filled to repletion with red blood-corpuscles (*engorgement*), and offer to the naked eye a red cuneiform spot. Some time after[1]—in the frog's tongue on the third or fourth day—as microscopical observation has shown, the red blood-corpuscles slip out through the uninjured capillary walls (by diapedesis) into the surrounding tissue. Thus arise small hemorrhages around the vessels, which constantly enlarge, coalesce, and present a real *infarction*. The *diapedesis of the blood-corpuscles is the result of a change not yet proven histologically in the walls of the capillaries, which must be considered as the effect of the cutting off of the supply of blood necessary for their nourishment.*

We learn, therefore, from this explanation, that not every embolus which gets into a branch of the pulmonary artery necessarily produces infarction; in a word, no such result follows

[1] This serves to explain the assertion made some time ago by Panum, that infarction does not immediately succeed the obstruction, but begins only after several days or hours.

when the artery in question is not a so-called "terminal artery" in the above-mentioned sense.

In many cases of pulmonary infarction, with mitral insufficiency and obstruction, the original thrombus is sought for in vain. Here we are obliged to suppose an anomaly of the walls of the vessels, which must be attributed partly to an increased blood-pressure, partly to imperfect nutrition, and consists very probably in a fatty degeneration of the finest arterioles. This change, going on from within outward, compromises the strength of the vessels to such a degree that an actual rupture takes place. Bleeding of this nature, according to Rindfleisch, is a quantitative exaggeration of those capillary hemorrhages which are seen under the same etiological circumstances in the so-called brown induration. The blood makes its way into a neighboring bronchus, and is then carried by aspiration into the corresponding pulmonary lobule and its final alveoli, and then, the blood continuing to flow, another bronchus is invaded, and the lobules belonging to it are filled, until finally the hemorrhage ceases by coagulation. According to Rindfleisch an infarction which has arisen in this way is distinguishable from one produced by embolism, inasmuch as in the former case the infarcted portion is very sharply defined from the surrounding healthy tissue, as a firm, uniformly dense, dark, bluish-red, wedge-shaped mass, while the latter fuses more gradually into the adjacent normal parenchyma through all the phases of hemorrhagic and of simple hyperæmia.

Metastatic abscesses of the lungs having a similar origin with embolic infarctions will be briefly mentioned here.

Since the researches of Virchow it is well known that metastatic abscesses are caused by emboli of a peculiar nature, which orginate from broken-down and infectious thrombi in the general venous circulation after operations, puerperal processes, etc. Their form readily distinguishes embolismal abscesses from infarctions, for the latter are wedge-shaped, the base being towards the periphery, the apex towards the hilus, while the former are more spherical or irregular. As regards situation, metastatic abscesses seem as a rule to have a very decided preference for the peripheral portions of the lung, although they do

occur in the middle and even at the hilus. Now these are not, like the hemorrhagic infarctions, the result of a simple mechanical process, but represent circumscribed foci of inflammation—*embolic pneumonia*—the consequence of obstruction of an artery by a plug, rendered infectious and capable of exciting inflammation probably by low organisms. The size of the inflammatory focus, and the longer or shorter time necessary for its purulent degeneration depend upon the size and specific nature of the obstruction as well as the reactive capacity of the surrounding tissue.

Pulmonary apoplexy, involving as a rule great destruction of tissue, arises without exception from the rupture of large, generally arterial, vessels, and rarely depends upon disturbances of nutrition in the walls of the pulmonary arteries—endarteritis and aneurismal changes—or upon their erosion by cancer, abscesses, and pulmonary gangrene. It is more frequently the result of injuries, gunshot and penetrating wounds, contusions and concussions of the thorax. Cases in which an aneurism of the aorta perforates a large bronchus may certainly offer symptoms similar to those of pulmonary apoplexy, namely, copious hemorrhages with suddenly fatal result; they are not, however, to be classified with the latter, for the pulmonary tissue generally remains intact. Only when the aneurism is adherent to the surface of the lung can its rupture cause extensive destruction of the parenchyma of the latter—pulmonary apoplexy. It is evident from the causes mentioned that persons in the prime of life are more liable than young people; and likewise, from predominance of traumatic causes, the male sex is more subject than the female. Ogston, out of twenty cases, reports fifteen males and five females: only two, out of the whole, were under twenty-one years of age.

Pathological Anatomy.

When death takes place soon after the occurrence of *bronchial hemorrhage*, blood, partly fluid and partly coagulated, is found in the air-passages, and may, by the aid of aspiration, extend into the finest bronchial ramifications and into the alveoli. The latter condition is especially marked in those cases in

which death has occurred from suffocation during the hemorrhage, because a considerable mass of blood escaping rapidly into the bronchi obstructs them, and is drawn inwards with a good deal of force during the struggles of the patient for breath. The lungs are then distended and overlap the heart, for, on account of the obstructing blood coagula, they cannot expel the air, and collapse. The lung is heavy, and its surface mottled with bright red spots, corresponding to the lobules which are the seat of hemorrhagic infiltration.

The mucous membrane of the bronchi is, in the majority of cases, reddened by imbibition and in a measure, according to what has been the fundamental cause, the seat of venous hyperæmia, relaxed, even softened, and not unfrequently studded with discrete or confluent extravasations of blood into the mucous and submucous tissues. The mucous membrane is found very anæmic, as are all other organs, only in those rare cases in which death has been caused by the loss of blood. In the majority of cases it is difficult to demonstrate post-mortem the source of even very copious bronchial hemorrhages.

The appearance presented by a *hemorrhagic infarction* is, as a rule, that of a wedge-shaped mass, the base turned towards the periphery of the lung and the apex, which is generally truncated, towards the hilus. Its *size* varies from that of a cherry or walnut to that of an apple, and occasionally may be such as to involve the third or the half of one lobe of the lung. As to *number*, there may be but a single infarction, though generally there are several. The smaller ones are found close to the periphery, as a rule directly under the pleura. The larger ones are situated nearer the centre or in the neighborhood of the root of the lung. They are relatively more frequent in the lower lobe, and in its most dependent portions, than in the middle and upper lobes. Those which are immediately beneath the pleura are distinctly recognizable from without by their dark-red, almost blackish-blue color, their hardness and projection above the surrounding pale and collapsed lung tissue, and, furthermore, by the fact that the pleura is at these points generally covered with a delicate layer of fibrine, the expression of a circumscribed pleuritis corresponding to the infarction. Some-

times, however, a rather copious sero-fibrinous exudation is found in one or both pleural cavities, the occasional red coloring of which is due to the escape of some blood from the vessels of that portion of the pleura covering the superficially located infarction. Upon section, also, the outlines of the infarction appear sharply defined from the normal parenchyma. The former is void of air, dark bluish-red, and resembles, according to Laënnec, a clot of venous blood; if pressure is made upon it, or if it is scraped with a scalpel, only a small quantity of a thick, half-coagulated, viscid, blackish-red liquid mingled with small granules exudes. The cut surface is smooth, though here and there it is granulated by reason of the fibrinous coagula contained in the alveoli and lobules. The tissue is at first elastic and firm, but later it becomes friable and inelastic. Spots occur here and there of a lighter hue, containing paler fibrinous coagula, as well as the whitish lines of vessels occluded by the white blood-corpuscles. The surrounding lung tissue is seldom entirely normal; it is generally hyperæmic, from collateral fluxion, and œdematous, and, if the infarction has been the result of serious mitral disease, is also very frequently the seat of brown induration.

On *microscopical examination* not only the vessels, alveoli, and bronchioles, but also the interstices, are found engorged with blood.

If the patient survives a long time, the infarction undergoes certain changes. The fluid portions are first absorbed, the blood-corpuscles disintegrate, and the fibrine undergoes fatty degeneration. The latter is then partly *absorbed*, partly *expectorated*, and the parenchyma of the lung, deeply pigmented by the coloring matter of the blood, may, if the infarction has been a small one, completely recover its normal condition, while if the infarction has been larger it *collapses* and *wastes*, or if reactive proliferation of the surrounding connective tissue takes place, it may form pigmented *pulmonary callosities*. Sometimes absorption is incomplete, and a yellow pulpy material remains which becomes *filled with calcareous salts*. Or else, the infarction having become liquefied and encapsulated, there remains a *cyst* of reddish fluid, sometimes containing crystals of

hæmatine. Sometimes, though rarely, inflammation of the adjacent tissue, with *formation of pus*, sets in and causes *sequestration* of the infarction, or, if secondary coagula form in the nutritive vessels of the neighborhood, even *gangrene*, with subsequent purulent or ichorous pleuritis. Even in these cases, under favoring circumstances, the sequestrated and gangrenous mass may become encapsulated and absorbed, granulations may form, and final cicatrization take place.

Metastatic abscesses occur, as a rule, in large numbers—a fact which is explained by the character, namely, the great friability and decomposition of the original thrombus material. They are situated both at the periphery, immediately beneath the pleura, and in the interior of the lungs. They are in general smaller than the hemorrhagic infarctions, and appear as round or irregularly-shaped masses the size of a pea or a cherry, which contain a thin, yellow, greenish, brownish, or blackish ichorous fluid mixed with detritus, and gradually fuse into the neighboring tissue, which is likewise discolored and infiltrated with fluid. Those which are peripheric project upon the surface of the lung. At these points the pleura is more widely affected and furnishes a purulent exudation, in consequence either of propagation of the inflammatory process to it, or of perforation with escape of the contents of the abscess into the pleural cavity. Metastatic abscesses are embolismal inflammations, resulting in the formation of pus and necrosis of the pulmonary tissue involved. Their whole course, according to Cohnheim, is identical with that of a genuine inflammatory pulmonary abscess, for the primarily red or grayish-red hepatization changes from gray to yellow, and goes on to purulent breaking down, which takes place either uniformly throughout the whole mass or gradually extends from the centre to the periphery.

Pulmonary apoplexy, which may occur in any lobe, attains generally a very considerable size, and is not sharply defined from the surrounding tissues, which are softened and infiltrated with blood. It forms foci filled with fluid and coagulated blood mixed with fragments and shreds of pulmonary substance, which, when superficially situated, or by extension to the surface, may perforate the pleura and discharge their con-

tents into the thoracic cavity (hæmatothorax). As a rule death occurs speedily, so that no further changes take place. Recovery by absorption of the effused blood, agglutination and shrinking of the parenchyma,[1] or by transformation into a serous cyst, is rare.

Symptomatology.

We shall not consider those minute traces of blood which, in the shape of little streaks and lines, are mixed with catarrhal expectoration as a consequence of very violent coughing—although they frequently alarm over-anxious patients, and cause them to seek medical advice at once—for they are, as a rule, of no significance and unassociated with any special symptoms; nor those more considerable quantities of blood—really bloody sputa—which are expectorated during chance attacks of coughing, after the inhalation of highly irritant gases, after severe bodily exertion, or during passive hyperæmia of the pulmonary circulation, and give rise to no further symptoms, and are likewise without danger. They disappear of themselves, and may reappear under similar circumstances.

True expectoration of blood—*hæmoptysis*—whereby very often considerable quantities of blood are raised, takes place not unfrequently when the patient is apparently in perfect health, while sitting still at table, or when making some very moderate bodily exertion, as walking, etc., without his having previously had a cough, or raised blood, or given any indication whatsoever of trouble in the organs of respiration. Sometimes, however, the hemorrhage occurs during severe exertions; such as dancing, gymnastic exercise, riding, running in the face of a sharp wind, loud speaking, shouting, singing, and the like.

Other persons—and they are generally those who have previously suffered from hæmoptysis, or who have a long, narrow chest, with a thin skin and great delicacy of the walls of the vessels, or whose lungs are already the seat of recognizable anatomical lesions—sometimes complain for a long period of congestion of the head, headache, dizziness, palpitation of the

[1] *Rokitansky*, Handbuch, 2. Aufl., Bd. III., p. 80.

heart, a feeling of oppression, and a sensation as if the breast would burst. Objective testimony is found in increased frequency and tension of the pulse (so-called *prodroma*).

These and like symptoms sometimes precede the actual hæmoptysis by several days, disappear after a short time to return again, or are of very short duration, and speedily followed by the hæmoptysis.

The attack occurs with the sensation of a warm fluid welling up from beneath the sternum into the throat, when by hawking the patient brings up several spoonfuls of almost pure blood having a peculiar sweetish or saltish taste. If the patient has not already coughed, a desire to do so is now felt, and more blood is raised with loud, moist râles, and a loose cough.

The blood is bright red, mixed with foam, though sometimes, especially towards the end of an attack, it is dark and coagulated, and its quantity is very variable. The hæmoptysis is frequently limited to two or three expectorations of a few spoonfuls, in other cases it continues for several hours, even half or a whole day, or, with interruptions, for several successive days, and the quantity of blood expectorated may amount to several pounds.

Small as the quantity of blood frequently is, it causes the patient nevertheless great anxiety and excitement. The pulse becomes frequent and tense. The face is red and burning. More frequently, especially if the hæmoptysis is somewhat copious, the patient becomes weak, the face covered with sweat, the pulse small and thready, and faintness, dimness of vision, and ringing in the ears set in. The patient faints, but soon recovers. The frequent pulse again becomes normal, provided some good reason for its remaining frequent (an eruption of tubercles and the like) is not present.

In most cases, for several days after the hemorrhage has ceased, dark-colored coagulated blood which has remained in the bronchi is raised by coughing and hawking, and then the hæmoptysis, according to the etiological forces and the condition of the lungs, may either terminate forever, or return again in the same way after a longer or shorter time. Sometimes it happens that a pretty copious hemorrhage is suddenly arrested,

and in a short time comes on again as profusely as before. This, according to Rasmussen, is because the opening in the vessel is occluded by a thrombus, which afterwards becomes detached and is forced through the same opening.

Interesting though it may be, when at the side of a patient suffering from hæmoptysis, whom one has never known before, to learn the condition of his lungs and the pathological changes which have caused the hemorrhage, yet in order to avoid excitement and renewed or more copious hemorrhage, a physical examination should for the time being be avoided. In the majority of cases in which hemorrhage has been moderate in quantity there is no dulness on percussion, for the blood, even when it has been respired into the air-cells, does not infiltrate the lobules to any great extent, and the reremain intermediate portions containing air. Auscultation reveals principally coarse and fine mucous râles caused by the blood in the bronchi. The infiltration is seldom so considerable that a more or less extensive dulness with bronchial breathing can be detected.

Every practitioner knows how frequently *hemorrhagic infarctions* of pretty considerable proportions are met with post-mortem, which during life were entirely unsuspected. True as it is that embolismal obstruction of large branches of the pulmonary artery gives rise to a sudden *dyspnœa*, threatening suffocation so that the patient is found with an expression of terror and despair, increased action of the heart, a small thready pulse, extreme cyanosis, cold extremities and chills, because a large portion of the pulmonary tissue is unavailable for the respiratory process on account of non-aeration of the blood in the capillary area of the occluded vessel, it is also true that when smaller branches of the pulmonary artery are obstructed this dyspnœa is entirely absent, or at least is not to be compared with that caused by mitral disease, with a want of compensation on the part of the heart. If, however, the infarction attain the size of a hen's egg, the sudden occurrence of great embarrassment in respiration, or the sudden augmentation of the existing dyspnœa is very striking. So long as these attacks are not repeated, the anxiety and dyspnœa gradually diminish, but return with

increased severity with every new infarction. This serves to explain the ameliorations and aggravations which are sometimes observed in the course of the disease.

The *characteristic expectoration* which is present in many cases is of great importance. This consists rarely of quantities of pure blood, but rather of an intimate union of mucus and blood, and has a certain resemblance to the pneumonic expectoration, though it is distinguishable from the rusty pneumonic sputum by being less tenacious and viscid, and by its dark-red, brownish, or violet-red color. The expectoration generally lasts several days, although the aggregate of blood[1] raised during that time is inconsiderable; it may even continue for several weeks.

Sometimes there are unmistakable symptoms of *pulmonary solidification;* this, however, can only be detected several days after the embolism has occurred, as is evident from the above description. Mixed mucous, followed by sibilant râles, and finally bronchial breathing, are audible over a limited extent of one lung, generally the postero-inferior portion. A solidification sufficiently extensive to be appreciated by percussion, and to cause an increased vocal fremitus, implies a relatively large infarction. These auscultatory symptoms will be sought for in vain if the bronchus leading to the hemorrhagic mass is occluded by a clot, for then only indefinite or very feeble and transmitted respiratory sounds are heard.

Under the same circumstances symptoms of *pleuritis* are also met with, such as a stitch in the side and a friction sound which at first is circumscribed.

It is almost unnecessary to state that the last-mentioned symptoms of pulmonary solidification and associated disease of the pleura are wanting in all cases of even pretty large infarctions situated near the hilus or in the centre of the lung, when they are covered by healthy lung tissue and do not extend to the periphery. Very frequently the dulness of an infarction situated in the lower portion of the lung is inappreciable because of its

[1] Laënnec (l.c., Meissner's German translation, I., p. 302) saw a young man raise ten pounds of blood within a period of forty-eight hours, after which he died; in less acute cases within fourteen days the quantity was about thirty pounds.

being concealed by a serous transudation (hydrothorax), the consequence of disordered circulation, or by a sero-fibrinous effusion. The dulness is thereby increased, and the above-mentioned auscultatory symptoms disappear.

A moderate degree of *fever* is exceptionally present, and is generally dependent upon secondary pneumonic and pleuritic affections. There are, however, cases reported (Penzoldt),[1] in which a moderate elevation of temperature—102°–103° F.—was present from the beginning. This agrees with the results of the experimental researches of Virchow, Bergmann and others, the latter of whom seeks an explanation for the rise of temperature in the increased muscular action induced by the intense dyspnœa.

In metastatic inflammations and abscesses caused by infectious emboli, the absence of prominent symptoms is even more conspicuous than in cases of hemorrhagic infarction. Dulness and abnormal respiratory sounds are entirely wanting, on account of the smallness of the foci. The characteristic expectoration is likewise absent in the majority of cases, because only a slight escape of blood takes place, and it is raised with difficulty, on account of the blunted sensibility of the bronchial mucous membrane, by the patients, who, apart from the hemorrhage, are suffering from serious illness. The existence of metastatic abscesses is to be suspected only in those patients in whom the etiological factors of such pulmonary affections are present, and who complain of chills and of pain in the breast, and raise reddish-colored, purulent, or thin brown sputa, and in whom auscultation detects friction sounds. In most cases their presence is only suspected or first realized upon the post-mortem table. If septicæmia sets in, or if it is already present, or if the

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[1] L.c., Cases 1 and 2.—He reports the case of a woman, sixty-nine years of age, with insufficiency and stenosis of the mitral valves, who was suddenly seized one evening with a severe chill, dyspnœa, and marked cyanosis of the face, and whose temperature the following morning was 102°. She died that evening. The autopsy showed that two-thirds of the right branch of the pulmonary artery was occluded by an embolus coming from the right heart, without any infarction or pleuritis having resulted up to that time. On the other hand, in the Jena clinic he saw cases with considerable pleuritic exudation following infarction, which ran their course entirely free from fever.

abscesses give rise by perforation to pneumo- or pyopneumothorax, then of course the symptoms of the case change.

Pulmonary apoplexy is characterized by profuse hæmoptysis. The blood sometimes pours out in streams from the mouth and nose, and as very frequently some of the blood is swallowed, hæmatemesis may occur. The patient in a short time becomes as pale as wax, and cold, the pulse small and frequent, the respiration hurried and superficial, and death, preceded sometimes by convulsive spasms, soon takes place, either from loss of blood, or, more frequently, from suffocation, in consequence of the bronchi becoming filled with blood. In the latter case, death may be caused so suddenly that but little or no blood is expectorated.

Duration, Termination, and Sequelæ.

The *duration* of bronchial hemorrhage is very variable. In most cases it is limited to a few inconsiderable expectorations; in others, it lasts from a quarter to a half hour, or even several hours, and then the attack comes to an end. More frequently, however, it recurs after several hours, or on the next day, and continues in this manner often for several days, or even one or two weeks, to come on anew at the end of perhaps several months or years.

If the total loss of blood has been relatively small, no material disturbance will result to the general system of the patient. If, on the other hand, a very considerable amount of blood has been lost, it will take a long time for the anæmic patient to recover entirely. Among the usual results from which the patient will suffer are shortness of breath, beating of the heart, ringing in the ears, spots before the eyes, fatigue upon the slightest exertion, slight attacks of fainting, etc. It is rare for the attack to end fatally by hemorrhage or suffocation.

Sylvius, Morton, Sydenham, Fr. Hoffmann, Boerhaave, Van Swieten, Andral, and others classed hæmoptysis among the causes of phthisis. This conclusion, however, yielded to Laënnec's assertion, that hæmoptysis is only a consequence of pulmonary disease, and never the cause of phthisis; and only very

lately F. v. Niemeyer, relying upon individual clinical observations, has again advocated with great enthusiasm the old opinion, and sought to still further strengthen it by theoretical evidence.[1] He affirms that after a hæmoptysis, occurring in previously healthy persons, the blood from the capillaries of the alveoli, or that which, after escaping from those of the bronchi, is drawn in by aspiration, gives rise in the lungs—just as a thrombus does in the walls of the blood-vessels—to an inflammation, the products of which, by disintegration and cheesy metamorphosis, cause phthisis in certain cases.

A case of this kind, upon which Niemeyer endeavors to base his assertion, came under the notice, among others, of Bamberger.[2] A gentleman, forty years of age, a member of a phthisical family, was suddenly, while in apparently perfect health, seized with an attack of hæmoptysis. In the course of a few days there was developed an acute infiltration involving almost the whole of the left upper lobe, followed by rapid breaking down, with the formation of cavities, to which, the pulse remaining frequent, was added an affection of the apex of the other lung. The patient died about six months afterwards. Niemeyer himself reports[3] the case of a young merchant in whom a pneumonic infiltration of the lower lobe of the left lung developed eight days after a severe attack of hæmoptysis. After the disease had continued for six weeks the patient died, and the autopsy revealed a cheesy infiltration of the whole lower lobe of the left lung, and also a cheesy wedge-shaped mass the size of an apple in the right lung.

A critical examination, however, seems to impugn the correctness of this very plausible proposition, founded upon such cases as the above. The perfect health of the patients is a very relative and subjective one, and every physician in large practice knows for how long a time persons with commencing phthisis go round with a slight cough which they think nothing of, and, regardless of all exposure, do not seek medical advice. The hæmoptysis first arouses them from their indifference, and in many cases, by close questioning, they will acknowledge that they have coughed for a long time, have even at times felt feverish and have lost strength. A physical examination then not unfrequently reveals an old, unsuspected focus of disease. Such

[1] *Niemeyer*, Handbuch.—Also his klinische Vorträge über Lungenschwindsucht, mitgetheilt von Dr. Ott, and Bürger's Dissertation.

[2] Würzburger med. Zeitschrift, 1861, Bd. II., p. 340.

[3] Berliner klin. Wochenschrift, 1869, p. 171.

cases as the above-mentioned admit, too, of another interpretation, in consequence of the careful observations of Buhl, whereby new light has been shed upon the different forms of pneumonia and their signification as factors in the production of phthisis. Buhl justly makes the distinction between catarrhal (superficial) and desquamative (parenchymatous) pneumonia, that the former never gives rise to extensive destruction of lung tissue, while the latter very frequently eventuates in phthisis. He also tells us that desquamative pneumonia, resulting in caseous degeneration, may, like croupous pneumonia, take place so very suddenly that it is sometimes found occupying a whole lobe, sometimes only a portion of one. Therefore the sudden change in the physical symptoms cannot be ascribed only to the hemorrhagic infiltration, for it may just as well be, and more frequently is, dependent upon the inflammatory infiltration of which the hemorrhage itself was a consequence. Moreover, cheesy degeneration never originates in catarrhal, but only in destructive desquamative pneumonia. Now, blood extravasated into the alveoli of the lungs only gives rise to catarrhal pneumonia, a fact which has been established by the experiments upon animals which have been instituted in different quarters for the elucidation of this question.

Perl and Lipmann, experimenting upon rabbits and dogs, opened the trachea and injected into the lungs several grammes of blood drawn from the jugular vein; examination at the end of twelve hours revealed the trachea, bronchi, and smaller branches of the latter, perfectly free from coagula, while the bronchioles and alveoli were the seat of brownish-red infiltrated foci which by the third day had decreased in size, and by the end of the fourth week had entirely disappeared, without there having been at any time the slightest evidence of inflammation of the parenchyma resulting in cheesy degeneration. After the experiments by Sommerbrodt upon dogs, consisting in the injection of partly fluid, partly coagulated blood into the trachea, microscopical examination showed—principally in the neighborhood of the hilus, and more in the lower than in the upper lobes, and on the right side than on the left—even at the end of three hours, besides abundant red corpuscles, certain changes in the alveolar epithelium, consisting in an increase in size, cloudiness, and the like, which were most marked on the fifth day, and after that gradually diminished, so that in the fifth week every trace of these changes had disappeared. Lebert has repeatedly seen large catarrhal cells as a consequence of pulmonary hemorrhages; and several years ago I endeavored to produce pneumonia artificially with various substances, but was never able by injecting blood to cause more than a

superficial catarrhal irritation. Waldenburg's [1] case proves nothing, for the cheesy hepatization found in the lower lobe of both lungs of a rabbit was rather the consequence of the abscess, which, as a result of the operation, formed in the subcutaneous tissue of the thorax and around the trachea, than of the blood injected into the trachea.

According to this explanation severe pulmonary hemorrhages in man, by causing accumulations of blood in the bronchioles and alveoli, give rise to a condition of superficial irritation which confines its action to the epithelium and may lead to caseous metamorphosis of the contents of the bronchioles and alveoli. This mass is, however, soon removed by absorption and expectoration, without causing any real destruction of the alveolar and bronchial walls, in other words, phthisis. We must therefore distinctly deny that bronchial hemorrhages may give rise to pulmonary phthisis. All those inflammatory symptoms mentioned by different authors, stitch in the side, increased frequency of the pulse, rise of temperature, and general indisposition, which are sometimes observed to accompany, or to come on a few days after, a hæmoptysis, are due to disease of the lungs, independent of the hæmoptysis, and have no further connection with the latter; this will account for our not having made mention of them in giving the symptomatology. The course and termination of this disease of the lung belongs therefore to another chapter.

From the anatomical description of a *hæmoptoic infarction*, its course and termination are readily conceived. It may undoubtedly heal—a result, however, which is less often observed than one would suppose. Even in favorable cases relapses must be expected, for the still existing cause, the thrombus, may at any moment furnish more emboli. If the process of infarction is frequently repeated, and a pretty considerable portion of lung tissue is thereby incapacitated for performing its part during respiration, the circulation, which has already been interfered with by the original trouble, will be still more embarrassed, and as a consequence the pulse becomes fast and small, the respiration quick and superficial, the patient becomes cyanotic, œdematous, somnolent, comatose, and thus gradually dies.

[1] Die Tuberculose. Berlin, 1869, p. 366.

Death is often caused, sooner or later, by the original disease and its consequences, especially in the case of organic disease of the valves of the heart. Not unfrequently, especially with large embolisms, death occurs very rapidly after the development of an infarction or before it has had time to form. Perforation of the pleura with pyo- or pyopneumothorax rarely occurs as the consequence of purulent inflammation or gangrene in the adjoining tissue.

Metastatic abscesses are, as a rule, followed by a fatal termination, on account of their original causes and their consequences, such as pyæmic infection, purulent or ichorous pleuritis, or pyopneumothorax.

Pulmonary apoplexy is invariably fatal from excessive hemorrhage or from suffocation, before medical assistance can be rendered. A less rapidly fatal result is only conceivable where the rupture of lung tissue is of proportionately limited extent.

Prognosis.

The prognosis of *bronchial hemorrhage*, so far as regards immediate danger, is, as a rule, favorable, for it is only in the rarest cases, and when the hemorrhage is very profuse, that the patient dies during the attack. Copious and frequently repeated hemorrhages may, however, by the anæmic condition which they induce, hasten the final dissolution.

In general, the prognosis depends upon the character of the original disease, upon the presence of extensive pulmonary lesions, and upon their nature. The presence of large cavities renders the prognosis most unfavorable, for then the bleeding, as a rule, comes from vessels situated in the wall, which are not completely occluded, and which, though for the moment perhaps obstructed by a thrombus, may, by the liquefaction or dislodgment of the latter, be the source of renewed hemorrhages. With regard to those bronchial hemorrhages which accompany or replace the menstrual flow, a most guarded prognosis should be given, for very frequently they are due to important lesions in the lungs. The most favorable prognosis is warranted when young, strong persons, who have hitherto enjoyed perfect health

and belong to a healthy family, are attacked with hæmoptysis as the result of some especial exposure.

We cannot agree with Lebert[1] when he asserts that a pretty copious hemorrhage during the early stage of pulmonary tuberculosis is relatively more favorable than the occurrence of several blood-tinged sputa associated with other signs of incipient phthisis, inasmuch as the former drains the affected lung tissue of blood, and thereby favors the shrinking up of the existing foci of disease; nor do we believe that pulmonary hemorrhages exert a salutary influence upon tubercular disease of the apices of the lungs. There is as yet no direct proof that hemorrhages have any such effect, and theoretical considerations are rather against than in favor of it, for a highly anæmic condition of any diseased part accelerates necrosis, and leads to destruction of tissue.

In *hemorrhagic infarction* the bleeding *per se*, on account of its small quantity, is of no importance to the patient, and therefore the prognosis depends entirely upon the primary disease (organic disease of the heart, emphysema, etc.) Nevertheless, extensive infarctions may, by diminishing the respiratory area, or by giving rise, though very rarely, to pleuritis and pyopneumothorax, hasten or directly cause the fatal termination.

Penzoldt[2] believes that a rather copious pleuritic exudation is an accident which favors the healing of an infarction, inasmuch as the pressure thereby exerted, acting like a compressive dressing, prevents further extravasation, and favors absorption of the infarction.

The prognosis of both *metastatic abscess* and *pulmonary apoplexy*, after what has already been said upon these subjects, requires no further consideration.

Diagnosis.

Whenever blood is raised by coughing or hawking, the physician should, by careful examination, endeavor to find the source whence it came, for it is not a very uncommon occurrence for hemorrhages from the nose, mouth, gums, throat, larynx, trachea, and also sometimes from the stomach, to be mistaken for bronchial and pulmonary hæmoptysis. During

[1] Klinik der Brustkrankheiten, II., p. 185.

[2] L. c., p. 30.

bleeding from the nose, if the patient lie upon the back, the blood may flow backwards and downwards into the larynx, where it excites coughing and supposed pulmonary hemorrhage; to avoid being deceived in such a case, one should learn whether bleeding from the nose has occurred upon that day, and should also carefully examine the nasal cavities. A thorough local inspection will also give the necessary information when the bleeding comes from the mouth or pharynx.

Especial care, however, is necessary here, for not unfrequently careless physicians are hasty in diagnosticating oral or pharyngeal hemorrhage, and are too ready to consider every dilated or tortuous blood-vessel which they may detect in these parts as the source of the hemorrhage. A special consideration of all the circumstances is requisite; the thoracic organs should be subjected to a rigid examination, and the question considered whether the local lesions present in the mucous membrane of the mouth and pharynx shall be looked upon as the *sole* source of the hemorrhage.

The laryngoscopical examination will, in the majority of cases, decide whether a hemorrhage is *laryngeal* or *tracheal*, for it then, almost without exception, is caused by new-growths, or by ulcers.

The discrimination between a hemorrhage from the lungs (hæmoptysis) and one from the *stomach* (hæmatemesis) is frequently more difficult, for the patient is very often unable to say whether the hemorrhage came on with coughing or with vomiting, or, since not unfrequently both occur, whether the cough or the vomiting set in first.

Although blood originating in the lungs is raised by coughing, and that coming from the stomach is necessarily accompanied by vomiting, yet it must not be forgotten that during the act of hæmatemesis some blood will get into the larynx and excite cough, and that during a severe hæmoptysis blood will be swallowed, and may give rise to vomiting. Moreover, the character of the blood, upon which great stress is very justly laid, will occasionally mislead one. Blood from the lungs is, as a rule, bright-red, frothy, and alkaline, and the clots formed by it are light, spongy, and filled with air-bubbles. That from the

stomach is, on the contrary, dark, even brownish or blackish, quite free from any air-bubbles, of an acid reaction, and occasionally mixed with bits of food, and the clots are firm and heavy. In those cases only in which the blood from the lungs flows from a large branch of the pulmonary artery, and is therefore heavily charged with carbonic acid, will it be of a similar dark color, and if poured forth in large quantities will likewise contain little air. On the other hand again, blood from the stomach, if it has escaped rapidly and in large quantities from a good-sized artery, and is immediately evacuated by emesis, will not have been long enough in contact with the fluids of the stomach to have been neutralized by them, and will therefore retain its bright red color and its alkaline reaction. In order to decide the question under such circumstances, other indications, which refer with more or less certainty to disease of the lungs or of the stomach, must be taken into account. For instance, on the one hand, the patient's constitution—perhaps a phthisical habit—the hereditary predisposition, a previous or existing cough, with emaciation, night sweats, etc.; on the other hand, symptoms dependent upon disorders of digestion, dyspeptic troubles, cardialgia, tumors located in the epigastrium, and also certain symptoms due to disturbances of the portal circulation.

It is frequently very difficult to determine positively whether a pulmonary hemorrhage comes from the capillaries of the bronchial mucous membrane or from a large vessel. In general, small hemorrhages are from the capillaries; nevertheless, judging by analogy from other mucous membranes, much larger hemorrhages may arise from the same source. It is only when hæmoptysis is violent, or when a physical examination reveals certain destructive processes, such as bronchial or parenchymatous cavities or abscesses in the lungs, that the hemorrhage arises probably from a large vessel.

I would call attention to one more source of error, which is not very uncommon. Certain hysterical ladies—and also other persons for the accomplishment of a fixed purpose—find it convenient to enlist the interest of those around them and of their physician by complaining of hæmoptysis, and by daily exhibiting their expectorations of blood. The ingeniousness of such patients is frequently marvellous, and the results of their endeavors often brilliant, especially if they are aided by accom-

plices. They sometimes deceive by filling the mouth with blood obtained by suction from the gums or from some other slightly wounded portion of the body, as the finger; more frequently, however, they endeavor to procure either the fresh or stale blood of animals, or certain substances which impart a red color. Several years ago I observed the case of an hysterical patient who procured a supply of old thickened animal blood, and daily prepared a certain quantity of it for her purpose by solution in water. The decomposed condition of the blood, and the absence of red blood-corpuscles, revealed by a microscopical examination, betrayed the fraud.

No one any longer agrees with the statement of Graves, that the blood raised from a hemorrhagic infarction can be distinguished from that belonging to a bronchial hemorrhage simply and solely by the color; that in the former case it is black and coagulated, in the latter, on the contrary, bright or dark red. To diagnosticate *hemorrhagic infarction*, its etiology must be borne in mind; seek for cardiac disease, with consequent dilatation, for pulmonary emphysema, or for thrombi in the peripheric veins of the body. If, moreover, a *high degree of dyspnœa* come on very suddenly, and cannot be referred to the primary disease, and if there is an *expectoration of mucus mixed with blood* associated with the sudden appearance of unmistakable physical signs of *circumscribed pulmonary solidification*, as well as some indications of *pleuritis*, the diagnosis of pulmonary infarction should be made without hesitation. This will be still further confirmed if the symptoms ascribed by Gerhardt to thrombosis of the right heart are also present, namely, a rather sudden increase in the lateral diameter of the heart, with irregularity of its action and that of the pulse, and disappearance of any cardiac murmurs, which latter, however, may at times alternately return and disappear, as occurred in a case reported by Hopf.

As we have already mentioned, many a case of hemorrhagic infarction wholly escapes diagnosis, and is only a post-mortem discovery, as, for instance, when it is of small size or is situated in the interior of the lung, or when the blood cannot be expectorated, owing to occlusion of the bronchial branch communicating with the infarction.

When the infarction is very large it may be mistaken for a croupous pneumonia occurring in a patient with heart disease, on account of the difficulty of breathing, the bloody sputa, the

dulness associated with sibilant râles and bronchial breathing. With an infarction, however, the sputa contain more pure blood and are fluid, not rusty colored, tenacious, and adhesive, and they continue to be present for a long time, while the pneumonic sputa retain this character for only a few days. A correct conclusion is also facilitated by bearing in mind the location of the dulness, which does not generally correspond with that of a pneumonia, its shape, and the fact that very frequently during pulmonary infarction several foci of circumscribed solidification succeed one another at intervals, causing repetitions and intermissions of the same symptoms.

Emboli of the *bronchial arteries*, with hemorrhage into and upon the bronchial mucous membrane, giving rise to similar symptoms—difficult respiration, bloody sputa, etc.—cannot be distinguished from Laënnec's central infarctions; the same may be said of the rare *thrombi of the pulmonary branches.*

The diagnosis of *metastatic abscesses* must, from what has already been said, as a rule, be a matter of great difficulty. It can be made with certainty if a patient who has been wounded, or has had a limb amputated, or is in the puerperal state, raises thin, limpid, brownish red-colored sputa, besides having the objective and subjective symptoms of pleuritis (friction sounds and pain in the side).

As a rule, the hemorrhage in *pulmonary apoplexy* is so profuse that death occurs before the arrival of a physician. Even if the latter find the patient alive, the impossibility of a thorough examination often renders the diagnosis of the origin of the hemorrhage, even as to whether it has come from the lungs or from the stomach, doubtful, which is a matter of little moment, however, in the treatment of such cases.

TREATMENT.

Bloodletting, by a single operation or repeated ones, the favorite means among the old-time physicians in the treatment of bronchial hemorrhage, has been, in recent times, more and more laid aside, because we possess means which are better and less injurious to the constitution. Where formerly it was the

custom to bleed from the foot, or apply leeches to the inner surfaces of the thighs and to the ankles, in the case of pulmonary hemorrhage following an absence of the menses, we now use other remedies if they are indicated, such as sinapisms to the thighs and loins, and stimulating lye or mustard foot-baths.

Bloodletting should be reserved for certain cases only; for instance those in which otherwise healthy and robust persons are attacked with intense pulmonary hyperæmia, tumultuous action of the heart, great oppression and sense of anxiety, followed by pulmonary hemorrhage, or, when in a case of serious passive hyperæmia from heart-disease, the stasis of blood in the lungs is proportionately very great when compared with the propulsive power of the heart. Oppolzer, moreover, advises a small venesection, from six to eight ounces, as an excellent means of combating hemorrhage in those cases in which the hæmoptysis has continued days and weeks in spite of all other remedies.

Whenever the hemorrhage is due to an altered condition of the blood, and consequent perverted nutrition of the walls of the vessels, the prophylaxis requires that special attention be paid to the patient's diet and mode of life. For in these, as in weak and slightly built patients, in those affected with tuberculosis and chronic inflammatory lesions of the lungs, such as precede phthisis, even a very slight exposure is sufficient to cause hyperæmia of the lungs followed by hæmoptysis.

These patients should be very careful to avoid every bodily exertion that is in the least degree fatiguing (dancing, riding, running), out-of-door walking when the weather is cold or the winds blow from the north or east, or during great heat; further, every mental excitement, hot and highly spiced foods and warm stimulating drinks, such as coffee, strong wines, and liquors. On the other hand, moderate out-of-door exercise during fine weather should be encouraged, and a residence in the country during the summer, and a warm climate for the winter and inclement seasons, recommended.

Whenever there is a well-marked *condition of anæmia*, *mild preparations of iron* are indicated, such as the lactate, phosphate, or reduced iron; also the internal and external use of *chalybeate waters*, bearing in mind the stimulating effects of the

carbonic acid, which in the case of those waters very rich in this gas may be modified by the application of heat or by the addition of milk or whey. The food should be nourishing and easily digestible, and the taking of milk should be particularly urged; special attention should also be paid to the regularity of the bowels.

During an *attack of hæmoptysis* complete *repose of the body* should be allowed the patient, best attained by the half-sitting position in bed, and all tight or uncomfortable articles of clothing should be removed. The patient should be strictly forbidden to speak, and all mental excitement should, as far as possible, be avoided. The room should be cool and airy, and the patient covered with a single covering; only cold food and drinks should be taken. To allay the *desire to cough*, *narcotics* are indicated, especially morphine or opium, and to regulate an excited action of the heart digitalis in infusion or tincture.

The most effective remedy for the *hæmoptysis* itself is *cold* applied externally, as an ice-bladder or cold wet compresses to the chest, and internally in the shape of bits of ice. Oppolzer recommends the gradual application of cold as more suitable, especially in the case of such patients as are very sensitive and dread the cold.

Among the customary remedies is the solution of chloride of iron,[1] taken internally, or better, by inhalation, diluted from twenty-five to one hundred times; another remedy is ergot in infusion (from nine to eighteen grains of ergot to four and a half ounces of water, one tablespoonful every one or two hours). It is less judicious to prescribe large doses in powder, because it irritates the throat and easily provokes a cough. In severe cases the aqueous extract of ergot (Bonjean) may be given subcutaneously. (Aqueous extract of ergot, two scruples; alcohol and glycerine, two drachms each:—of this inject from one-half to one syringeful.) Worthy of mention, moreover, are tannin and gallic acid (Waters), nine grains every one, two, or four hours; alum, four and a half grains every two or three hours, in less severe cases an alum whey by the cupful. Rogers recom-

[1] "Strong solution of perchloride of iron."—*British Ph.*

mends the tannate of alumina, and as a more powerful remedy, ferric alum. Furthermore, acetate of lead, best combined with a grain of opium, every hour or half hour. The *balsams*, especially oil of turpentine, five, ten, or twenty drops in gelatine capsules; the copaiba, best in the *potio Choparti* as modified by Wolff[1] (copaiba, syrup of tolu, peppermint water, alcohol, each an ounce; spirit of nitrous ether, thirty minims, in teaspoonful doses). The *mineral acids* are frequently given, especially in mild cases, such as the dilute sulphuric or phosphoric acids, or Haller's acid elixir,[2] from ten to fifteen drops in a mucilaginous vehicle every two hours; occasionally combined with tincture of digitalis if the action of the heart is very excited, and if there is much cough, with bitter almond water or with morphine.

A popular remedy, and indeed one of some efficacy, and always at hand, is *common salt*, which in case the physician and the more appropriate medicines cannot be had at once, should be given in doses of from a half to one tablespoonful, either dry or mixed with a little water. The effect, according to Scoda, is caused by the salt acting as an irritant upon the gastric mucous membrane, producing through the sensory nerves of the stomach a reflex action upon the small pulmonary arteries, whereby the latter are contracted.

The administration of *emetics* in hemorrhages, especially ipecac in nauseating doses, so highly recommended by Graves and Trousseau, has lately received new advocates (Peter, Massina, H. Weber). Peter refers the effect to the diminution in the size and strength of the pulse, and Weber suggests that, besides this, the expulsion of the blood from the bronchi, where it has accumulated and become a source of inflammation, is thus facilitated. There is really nothing to be feared from the blood thus accumulated, and the effect of ipecacuanha upon the heart and vascular symptom is as yet uncertain and but little understood. On the other hand, the danger that new hemorrhages may be produced by the jarring of the body during the act of vomiting, is so apparent that this mode of treatment should meet

[1] Charitéannalen, 1852, Bd. II., 2.

[2] Pure sulphuric acid, 1 part: add by drops while stirring to alcohol, 3 parts.—*German Pharmacopœia.*

with disapprobation in spite of the high standing of its advocates.

If the bronchial hemorrhage merely consists in the daily expectoration, after coughing or hawking by the patient, of two or three bloody sputa, or in the raising of mucus streaked with blood every morning, a simple dietetic treatment is all-sufficient, the avoidance of all bodily exertion, of hot and exciting food, etc., and no special medication is required. But, out of regard for the patient's anxiety, it will frequently be necessary to do something, and then the mineral acids may be prescribed.

Notwithstanding Laënnec's advice, that in *hemorrhagic infarction* the bleeding should be combated during the first or second day by "copious" bloodletting—from twenty to twenty-four ounces—this remedy is no longer employed so freely. Venesection in infarction is not indicated to subdue the hemorrhage, but is of use only where there is an intense degree of passive congestion in the pulmonary circulation threatening œdema of the lungs.

In many cases where there is increased frequency of the heart's action, with incomplete contraction of that organ, acids, and more especially *digitalis*, are indicated. With a little precaution, and particularly by reserving this latter remedy for the urgent cases, the danger feared by Gerhardt and Penzoldt, that it favors thrombosis of the right side of the heart and gives rise to new emboli, may be avoided.

In many cases embolism of a large vascular branch is quickly followed by collapse, when *stimulants* should be administered, such as wine, camphor, musk, and the preparations of ammonia internally and externally, hand- and foot-baths, embrocations with the spirit of mustard,[1] mustard poultices to the breast and the inner aspect of the thighs.

If the hemorrhage is considerable in quantity and requires special treatment, the indications are the same as those mentioned for bronchial hemorrhage.

It is impracticable in the case of *metastatic abscesses* to make use of any special treatment, therefore the latter should

[1] Oil of mustard, 1 part; alcohol, 50 parts.—*German Ph.*

coincide with that of the primary disease. For *pulmonary apoplexy*, if the physician arrive in time, cold should be made use of externally and internally, the subcutaneous injection of ergotine, and internally, perhaps, the solution of chloride of iron. If collapse threaten, analeptic remedies should be given. Furthermore, all those indications are applicable which have already been enumerated in the treatment of bronchial hemorrhage.

ATELECTASIS.

HISTORY AND LITERATURE.

Atelectasis, although long ago observed and described as it occurs in new-born infants, was looked upon by former writers, and especially by French authors (*Valleix*, *Billard*, *Grisolle*), as a congenital pneumonia. *E. Jörg*, in the year 1832, in his treatise: "De morbo pulmonum organico ex respiratione neonatorum imperfecta orto, Lipsiæ, 1832," and later, in den Analekten der Kinderkrankheiten, Stuttgart, 1835, Heft, V., and also in a special work: "Die Foetuslunge im geborenen Kinde für Pathol., Therap. u. gerichtl. Arznei-Wissensch., Grimma, 1835," was the first to describe this condition as a residue of fetal life and as the result of imperfect respiration, that is, as an incomplete expansion of the lungs with air. He describes this in the above-mentioned treatise, p. 6, in the following happy manner: "Hic constat concretione cellularum pulmonalium, quæ majorem vel minorem partem dextri vel sinistri, vel utriusque pulmonis comprehendit. Cellulæ vero istæ nulla præcedente inflammatione, nullaque exsudatione sive destructione sequente, solummodo ex aëris ad illas propter respirationem incompletam ac superficialem non penetrantis, defectu, sensim sensimque conglutinantur denique re vera coallescunt."

This anatomical description by *Jörg* was supplemented by the investigation of several cases by *Hasse* (Spec. pathol. Anat. Leipz., 1841), who pointed out the differential anatomical features between atelectasis and certain inflammatory conditions. Then came the descriptions of *Legendre* and *Bailly* (Archiv, gén., 1844, Janv.-Mars): "qu'il n'y a point de travail de décomposition et formation des produits pathologiques mais seulement modification physique dans la structure des organes." These investigators also observed the same conditions in older children, and declared that atelectasis, as found in new-born infants, is not, as *Jörg* and *Hasse* say it is, a remnant of the fetal state, but a retrogression to that condition.—*Mendelssohn* (Der Mechanismus der Respiration und Circulation u. s. w. Berlin, 1845, p. 177 et seq.) endeavored to produce atelectasis of one lung in animals experimentally, by opening the corresponding

pleural cavity. If the animal died soon after the operation, the lung could be inflated, which, however, could not be done if death only occurred after a long time. He declared atelectasis and pneumonia of early childhood to be different degrees of development of one and the same process, inasmuch as atelectasis gives rise to a condition of stasis in the capillaries, which may be followed by exudation and the development of plastic material, and then pneumonia.—*Traube* (Beiträge zur experim. Pathol. u. Physiol. Berlin, 1846, Heft I., p. 184 et seq.) described atelectasis as it occurs in adults. The clinical symptoms corresponding to the anatomical changes have been accurately described, and the differential diagnosis of these two conditions, atelectasis and pneumonia, so often confounded the one with the other, established by *Friedleben* (Archiv für physiol. Heilkunde, 1847, Jahrg. VI.); *Rees* (Atelectasis pulmonum, or, Closure of the Air-Cells of the Lungs in Children. Lond., 1850, 42); *Donders* (Nederl. Lancet, 1851, Mei); *West* (Lecture on the Diseases of Infancy and Childhood, translated into German by Wegner. Berlin, 1853); *Cohn* (Günsburg's Zeitschr., 1854, Band V.); *Graily Hewitt* (On the Pathol. of Whooping-Cough. Lond., 1855. Diagnosis of Apneumatosis (Pulmonary Collapse). Brit. Med. Journ., Nr. 15, and Lancet, Nr. 25, 1857); *Gerhardt* (Beiträge zur Lehre von der erworbenen Lungenatelektase. Diss. Würzb., 1857, and Virchow's Archiv, 1857, Band XI., p. 240); *Bartels* (Virch. Archiv, 1861, Band XXI., pp. 65 and 129.

Berg, Aus dem Jahresber. über das Stockh. Waisenhaus, Journ. für Kinderkrankh., 1846, Juli; *Legendre*, Recherch. anat. path. et cliniq. sur quelques maladies de l'enfance. Paris, 1846, p. 159 et seq.; *Eulenberg*, Preuss. Vereinszeitung, 1848, Nrs. 6–8; *Köstlin*, Arch. für phys. Heilkunde, 1849, Jahrg. VIII., 2, 3, 6, 7; 1854, Jahrg. XIII.; *Gaildner*, Edinb. Month. Journ., 1850, Vol. XII.; *Forsyth Meigs*, Amer. Journ., 1852, Jan.; *Baron*, Journ. für Kinderkrankh., 1852, 18; *Rühle*, Lungencollapsus, 32, Jahresbericht der Schlesis. Gesellschaft, 1854; *Günsburg*, Klinik der Kreislaufs- und Athmungsorg. Breslau, 1856, p. 476; *Radetzky*, Die Pathol. der katarrh. Lungenentzündung der Neugeb. u. Säuglinge. Diss. Petersb., 1861. Petersb. med. Zeitsch., Band I., p. 147; *Ziemssen*, Pleuritis und Pneumonie im Kindesalter. Berl., 1862, p. 296; *Thomas*, Nederl. Tydschr. v. Geneesk, 1864, Juni, Band VIII.; *Steffen*, Klinik der Kinderkrankh. Berl., 1865, Band I., p. 45 et seq.; *Cockle*, Acquired Atelectasis of the entire Upper Lobe of the Right Lung from direct Mech. Pressure. Assoc. Med. Journ., Nr. 204, 1856; *Biermer*, Handb. der spec. Path. u. Therap., 1867, Band V., 1. Abth., 5. Lieferung, p. 819.

Compare also the anat. plates of *Cruveilhier*, Anat. pathol. Livr., XV., pl. 2; *Albers*, Atlas der path. Anat., Band III., Taf. 25; *Casper*, Atlas z. Handb. d. gerichtl. Med., Taf. VI.; likewise the text-books on path. anatomy by *Rokitansky*, 3. Aufl. Wien, 1861, Band III., p. 58; *Förster*, 2. Aufl. Leipz., 1863, p. 240, etc., and *Weber*, Beiträge zur path. Anat. der Neugeb. Kiel, 1852, 2. Lieferung, p. 34 et seq.; also the text-books upon the diseases of children by *Friedberg*, *Billard*, *Bouchut*, *Rilliet* et *Barthez*, *Bednar*, *Henning*, *Vogel* and *Gerhardt*.

DEFINITION.

Atelectasis (ἀτελής, imperfect, and ἔκτασις, dilatation) or *apneumatosis* are names given to that condition of the lungs in which expansion of more or less of the lung is imperfect or entirely absent, and when this lack of expansion is not due to the filling of the alveoli with fluid or with a coagulable material, but depends wholly upon the small capacity of the alveoli which may even amount to direct apposition of their walls the one with the other. Although this condition is normal in fetal life, yet it becomes pathological whenever this lack of expansion continues after birth. It then constitutes a *congenital atelectasis*, or *atelectasis in its narrower sense*, or état fœtal (Legendre). A portion of the lung, however, which has been perfectly expanded, may also, later in life, lose its capacity for air. This takes place, as a rule, by a sort of resorbing process in those portions of the lung which are shut off by the occlusion of their bronchi. The walls of the alveoli collapse and lie against one another as in the fetal state; this is *acquired atelectasis*, *obstructive atelectasis*, or *collapse of the lung*. The effect of external pressure upon the lung may drive the air out from the alveoli and lobes, and give rise to *atelectasis from compression*.

ETIOLOGY.

Congenital atelectasis is most frequently met with in feeble children, those who have been born prematurely, or who have come into the world in a state of apparent death after a difficult or tedious labor, and is due to unequal and imperfect action of the respiratory muscles. It occurs, moreover, in those children whose bronchi are partially obstructed by mucus or meconium.

Collapse of the lungs takes place most frequently during childhood, especially during the first weeks and months of life, less often among adult persons, and is also due to occlusion of small bronchi by tenacious mucus, dependent upon a chronic catarrh of the terminal bronchi, which, by reason of their small calibre and the thinness of their walls, are readily sealed up and obstructed. Besides the catarrh and its consequences, the

strength of the respiratory muscles must be taken into account, for in proportion as this is below the normal standard, will they be unable to overcome the obstacles placed in the bronchi by the catarrh to the entrance of air into the alveoli. The respiratory force depends, according to Gerhardt, upon the age of the child, upon its general nutrition, especially upon its muscular development and degree of innervation. Therefore, we meet with atelectasis both in very young infants and in those children whose general nutrition is very much reduced by other diseases, such as chronic diarrhœa, rachitis, whooping-cough, measles, or by poor hygienic influences, bad air and improper food. That diminished innervation may cause atelectasis, is shown by cases of brain disease, coma, and sopor in children, and by severe cases of typhus in adults. The practice of swathing children, which has often been urged as a cause, is without influence upon the origin of atelectasis. The same may be said of the following conditions, which have likewise been termed etiological: the respiration of a dusty and vitiated atmosphere, and long-continued dorsal decubitus of a child, factors which are of importance only so far as they favor pulmonary hyperæmia and catarrh, and thus indirectly induce atelectasis. According to Gerhardt, collapse of the lung may occasionally be caused by extensive pleuritic adhesions. It is now and then produced by paralysis of the pneumogastric nerve, or by the obstruction of a bronchus by a neoplasm of the lungs, or by the presence of a foreign body. Collapse of the lung is still more rarely the result of compression of a bronchus by external pressure, whereby, in proportion to the size of the bronchial branch, atelectasis of a larger or smaller portion of the lung is produced.

Cockle reports an interesting case of this kind, in which the entire upper lobe of one lung became the seat of atelectasis in consequence of pressure exerted upon the right bronchus by enlarged bronchial glands. The rest of the lung was emphysematous, and contained scattered miliary tubercles. The right auricle was considerably dilated, the left ventricle dilated and hypertrophied, and the foramen ovale patulous.

To correctly appreciate the disappearance, that is, the absorption, of the air from the lobules in ordinary cases of collapse of the lung, it is necessary to remember that during inspiration the

elasticity and contractility of the parenchyma of the lung are opposed to, and must be overcome by, the action of the respiratory muscles. This resistance is increased by the undoubtedly greater elasticity of a child's lung, and also by the accumulation of mucus in the bronchi, in consequence of a catarrhal condition, and still further, as Bartels believes, by contraction of the bronchial muscles, brought about through reflex action by the irritated catarrhal mucous membrane. If, now, in addition, the inspiratory muscles are weak, and there is deficient innervation, one can readily conceive that no air can get into the alveoli from without. The air which they already contain, and which, by reason of the occluded bronchi, is shut in, is subjected to high pressure by the elasticity and contractility of the alveolar parietes, and is absorbed by the blood, as was first pointed out by Virchow.

Atelectasis from compression is caused by the pressure of fluid in the pleural sacs; by pleuritic effusions, empyema, and hydrothorax; also by pneumothorax, of which we can most easily convince ourselves by opening a pleural cavity in an animal. This condition is also found with an accumulation of fluid in the pericardium, hypertrophy of the heart, tumors in the thorax, aneurisms, large tumors in the lungs, distortions of the spine, rachitical deformity of the thorax, fluid effusions and large tumors in the abdominal cavity, as well as a high degree of tympanites, whereby the diaphragm is pressed upwards.

Any of these conditions may compromise more or less pulmonary tissue, and cause it to undergo atelectasis.

Pathological Anatomy.

Congenital atelectasis is most frequently situated in the posterior and inferior portions of the lungs, less frequently in the anterior borders, the tongue-shaped processes and the apices of the lungs. It occurs either as a single circumscribed lobular mass, or it may, though less frequently, involve considerable portions of lung tissue, the half, or even the whole of one lobe.

These atelectatic portions are sunken below the level of the adjacent normal lung tissue. Superficially they are violet, red-

dish-blue, or steel-colored, while the interior as seen upon section is more brownish-red. The pleura covering them is perfectly normal. The tissue is dense, and does not crepitate upon pressure or when cut into. It is tough, not easily torn, and between the fingers feels soft and flaccid, and not at all friable. The free edges of the lungs, owing to imperfect expansion of the parenchyma at these points, are shortened and turned inward. Small pieces of lung tissue sink in water. Sections of these portions appear smooth, not granular, void of air, condensed, have but little fluid constituents, and only a slight amount of blood can be pressed out. At first the portions of lung thus affected are entirely inflatable, and may thereby regain their normal pale-red color and the other characteristics of healthy lung tissue; but after some time this is no longer possible. The portions involved become denser and harder, contain less blood, and are drier. The changes which under these conditions take place in the lung substance, whereby dilatation of the alveoli by insufflation of the lung is rendered impossible, have not yet been satisfactorily determined. That the walls of the alveoli, according to Jörg and others, become agglutinated, and eventually grow together, is more than doubtful. According to Rokitansky, there is demonstrable, besides a fatty degeneration of the alveolar epithelium, an abundant cell-proliferation in the tissue, which is followed by a permanent growth of connective tissue, with destruction of the pulmonary substance. We do not, however, believe that this process is, as Rokitansky thinks, dependent upon a form of inflammation due to abnormal conditions of diffusion. That a sort of tissue-metamorphosis takes place, and that there is not a simple juxtaposition of the alveolar walls, is shown by the fact that during fetal existence the lungs are collapsed, but do not thereby undergo such changes that inflation is no longer possible.

In *acquired collapse of the lung* the anatomical conditions are in general of a similar kind, though the atelectatic points are more disseminated and scattered throughout both lungs; they show a preference for a lobular arrangement, and correspond more closely with the distribution of the bronchi, to the occlusion of which they owe their formation. They have in slight

cases a superficial location, but they may, by extension, involve the deeper parts of the parenchyma. In very severe cases the apneumatic portions extend over large sections of the lung, and, as observed by Bartels and Steffen, sometimes stretch in a band of more than an inch in width, upon the posterior surface of the lung, from the apex to the base. Their color is generally a dark steel-blue, owing to engorgement, and this feature is frequently all the more striking from the fact that the lobules of the rest of the parenchyma of the lung are often bloated and of a pale color, from the coexistent, vicarious, vesicular emphysema. The cut surface is less dry, less poor in blood and serum, and, owing to the existing catarrhal condition, a blood-red, viscid serosity can be scraped from it. If a passive or hypostatic hyperæmia is associated with the collapse, the cut surface seems softened, is of a dark-blue color, and blood can be freely expressed. From the close resemblance of the parenchyma to a section of the spleen, this condition has been called *splenisatio pulmonum.* The pleura is normal, or may occasionally be the seat of a few ecchymoses; only in rare cases is it thickened or retracted.

The bronchi are considerably congested, their mucous membrane swollen, and it is not unusual to find in them yellow occluding plugs of mucus; in addition to the atelectatic portions, pneumonic infiltrations are frequently met with.

When *compression* has caused the atelectasis, the location and extent of the apneumatic portions depend upon the causative agent. They occur most frequently at the base and in the sharp edges of the lungs, often involving large portions of a lung, or a whole lobe, very rarely the whole organ. Thus, compression of the base of the lung is produced by pressure upwards of the diaphragm, owing to distention or filling of the abdominal cavity; in skoliosis the posterior portions of the lungs are affected, and, according to Rokitansky, on the side of the convexity of the spinal curvature, corresponding to the fissure-like shape of the posterior thoracic cavity. With pericardial effusion the anterior borders of the lungs suffer; with free pleuritic effusions, the lower portions; while with sacculated circumscribed pleuritic effusions, those portions corresponding to the position

of the fluid collections. The degree of compression is variable. The lung is either simply diminished in size and rendered somewhat more compact, but still retains a portion of its air, or it is perfectly void of air, though the vessels still remain filled with blood, or else the lung is compressed to such a degree that both air and blood are driven out. As long as the tissue still retains its blood supply, it is dark-red, and resembles upon section a piece of muscle—*carnificatio pulmonum*;—while if the blood has been completely expressed from the lung, its color is brownish or bluish, most frequently, however, grayish-blue, and finally uniformly gray. The parenchyma is then dry, perfectly empty of air, tough and leathery. The texture of the lung proper and the small bronchi disappear, and finally nothing remains but a cicatricial mass of connective tissue, with thickening of the pleura. In such a condition the lung tissue can no longer be inflated, and of course during the remainder of the patient's life is unavailable for respiration.

Though the anatomical changes in atelectasis are very characteristic, yet it not unfrequently happens that those of limited experience confound them with certain inflammatory conditions, that is, with pneumonic infiltrations, and it will therefore be fitting to enumerate here the most important distinguishing features. Atelectatic portions of the lung are sunken below the surrounding normal lung tissue, and are of less extent than portions that are the seat of pneumonic infiltration. The cut surface is smooth, non-granular, and no croupous or cellular exudation can be pressed out. Further, the atelectatic portions can be inflated for a long time by insufflation through the trachea, which is impossible with an inflammatory infiltration. The atelectatic tissue, moreover, is firm, leathery, non-friable, and the pleura quite smooth and free from pseudo-membranes.

Patulousness of the fetal openings is a not infrequent *consequence* of extensive congenital atelectasis. The foramen ovale and the ductus arteriosus Botalli remain patulous, as a result of the influence of disordered respiration upon the circulatory system. The healthy lung during inspiration facilitates by its suction power the emptying of the right heart, and during expiration, by its contraction, assists the discharge of the blood from

the lung into the left auricle, and onward into the general circulation. In atelectasis, when this suction and propulsive power is very materially diminished, the right ventricle becomes engorged with blood, and will, during contraction, force a portion of the latter from the pulmonary artery through the ductus arteriosus Botalli into the aorta, and again some blood will flow from the right auricle through the foramen ovale into the left auricle, preventing, very naturally, the closure of these openings. It is therefore very apparent that a catarrh followed by pulmonary collapse during very early infancy may be a cause of imperfect closure of the fetal openings.

Atelectasis, both in children and adults, occasionally results in lobular pneumonia. This of course is only possible while the alveoli are still permeable for air, and also therefore for an exudation.

Symptoms and Course.

An atelectasis which is circumscribed and of small extent will cause no symptoms whatever; it is only when larger portions of lung tissue are the seat of this condition that during life it gives tokens of its existence and can be recognized by the physician.

The symptoms of *congenital atelectasis* are, as a rule, present from the moment of birth, although during the following days they become more pronounced and no longer admit of any doubt as to the nature of the disease. The symptoms are above all those of *insufficient respiration.* The breathing is superficial and rapid, the thorax expands inadequately, the voice is weak, small, and finally inaudible; the children do not cry, but only whimper, and are unable to nurse with any vigor.

The little patients become cyanotic from the imperfect performance of respiration and from the afore-mentioned patulous state of the fetal openings. The lips and terminal phalanges are blue, the face livid, and the surface of the body pale and cool. They become drowsy and sleep a great deal, the extremities are moved very slowly, and generally remain relaxed and motionless. The bowels are quiescent, and no meconium is passed. Slight muscular twitchings, especially in the face, are often observable, and convulsions, contractions, and paralyses

are not infrequent. The passive hyperæmia resulting from the *anomalies of circulation* may give rise to the formation of blood-clots in the cranial sinuses, three cases of which have been observed by Gerhardt. The diagnosis of this complication is determined by drowsiness, slight facial hemiplegia, and unequal distention of the jugular veins, one of them being enormously engorged, while the other is in a state of utter collapse.

In severe cases death generally occurs within a few days after birth, sometimes not until the third or fourth week, and rarely at a somewhat later period, with the phenomena of extreme debility and atrophy. Now and then the child will die with all the signs of suffocation from excessive carbonization of the blood caused by the patency of the fetal openings, or in an attack of convulsions.

Whenever congenital atelectasis does not end fatally within a short time, the symptoms do not differ from those of *collapse of the lung*. Both conditions present the features of insufficient respiration and imperfect decarbonization, and certain disturbances of the circulatory and nervous systems immediately dependent upon them. The bronchitis, however, to which the collapse of the lung is secondary, may also give rise to the same symptoms in the children, for it causes insufficient interchange of gases and dyspnœa, so that unless one is aided by physical signs the diagnosis is frequently difficult. In acquired atelectasis respiration is labored, arduous, and of increased frequency, the thorax is expanded with an effort, and unequally, if the disease is confined to one lung.

The manner of respiration undergoes a peculiar characteristic change, which was first noted by Gerhardt, and later by Graily Hewitt, similar to that which occurs in croup and in laryngo- and tracheostenosis. For instance, when, during the act of inspiration, the thorax expands by an elevation of its upper portion, and, by contraction of the diaphragm, the lungs, being partially impervious to air by reason of the atelectasis, are unable to accompany this expansion. Therefore, a compensating retraction of the thorax takes place at its most pliable points, recognizable partly by the sinking in of the intercostal spaces, and partly by a retraction of the sternum and the lower

true and false costal cartilages. The more yielding the thorax, the more striking is this symptom.

Physical examination only yields positive signs when the atelectasis is moderately extensive, involving, according to Gerhardt, an eighth or a sixth of one lung, and when the individual points of disease are not too far from one another.

The inferior boundaries of the lung, both anteriorly and posteriorly, remain normal, because the atelectatic portion is generally compensated for by an expansion of the normal lung tissue (emphysema) and by the narrowing of the thorax. The præcordial dulness is, as a rule, increased in extent, which is explained partly by the fact that the heart is less covered by the lungs, and partly by a dilatation and hypertrophy of the right side of the heart, in consequence of disorders in the pulmonary circulation. Quite in accordance with the latter symptom is the larger surface over which the heart's beat is appreciable, as well as its increased force. When the atelectasis has caused considerable condensation, pretty distinct dulness can be detected over the corresponding point by light percussion.

On auscultation the respiratory sounds may be entirely absent, because no air can enter that portion of the lung which is the seat of atelectasis. Only a gentle vesicular breathing can be heard, which is transmitted from the neighborhood, and may be mingled with rhonchi if catarrh also is present. When the atelectatic process is very extensive, bronchial breathing and bronchophony, with increased vocal fremitus and consonant rhonchi, are appreciable.

The cough which is frequently present is due to the catarrh, and all feverish symptoms should be ascribed not to the atelectasis, but to the complications, viz., the original disease, repeated catarrhal attacks, or a concomitant broncho-pneumonia. The rapid action of the heart is owing to the disorders in the pulmonary circulation.

Many of the above-mentioned symptoms, which are very significant in congenital atelectasis and in collapse of the lung, are also, for very apparent reasons, present in compression of the lung. The symptoms likewise of atelectasis from compression sometimes coincide with those of the original disease, or

are obscured by the latter. When the compressed lung is still to a certain extent permeable to air, the percussion sound is tympanitic, while if the air has been completely driven out, percussion gives a flat sound. If, on removal of the cause, air re-enter a portion of lung substance which has been greatly compressed, the percussion sound becomes gradually tympanitic and finally normal.

Among the other complications depending upon an imperfect discharge of blood from the right heart, and which are the more striking in proportion as the atelectasis is more extensive, should be reckoned in addition to the already-mentioned cardiac symptoms, the backing-up of the blood in the general venous circulation, in the brain, liver, kidneys, inferior extremities, etc. This condition gives rise to passive hyperæmia and œdema and their associate symptoms, and occurs most frequently in connection with very extensive atelectasis from compression. In consequence of the insufficient filling of the left heart and of the aorta, the pulse becomes small, and the skin pale and cool ; the urine is scantily secreted, has generally a high specific gravity, is dark colored, and has an abundant uric acid sediment. A condition of active hyperæmia (fluxion) may exist in the non-atelectatic portions of the lung, and if it occur suddenly, or if the atelectasis from compression should extend considerably, pulmonary œdema may supervene.

Prognosis.

The prognosis of congenital atelectasis depends upon the amount of lung involved, and upon the nutritive condition and strength of the child. In extensive atelectasis in very feeble children it is unfavorable ; but in strong, otherwise healthy children it is favorable, provided the external surroundings and the other conditions of life of the little patients are all that can be desired, that is, provided pure and fresh air, a healthy residence, proper food, suitable warm clothing, in a word, careful nursing, are insured. Under such conditions, as the child grows older, the lungs seem to become perfectly expanded ; nevertheless, many authorities state that children who have suffered in

early infancy from atelectasis retain a certain feebleness of the respiratory organs, and suffer very frequently from hyperæmic and catarrhal conditions of the lungs. This assertion is, however, more than doubtful.

In collapse of the lung the prognosis depends likewise upon the extent of the atelectasis, the degree of the patient's strength, the above-mentioned favorable or unfavorable surrounding circumstances, and the primary disease. Even in this case, however, an appropriate and early instituted treatment will render the result still more favorable. If the original disease can be removed, and the atelectasis has not become too extensive or existed too long a time, the prognosis is not unfavorable. According to Gerhardt, the degree and the extent of the morbid process, provided the same is not associated with some other thoracic or laryngeal disease, should be estimated by the amount of the compensating contraction of the thorax.

The prognosis of atelectasis from compression depends upon the causative disease, whether this can be entirely and speedily removed, and also upon the duration and extent of the compression.

Diagnosis.

If careful attention be given to the individual symptoms, the diagnosis offers no special difficulty; still, now and then, this affection is mistaken for *bronchitis*, *pneumonia*, and *liquid effusions* in the thorax. The diagnosis from bronchitis, which may likewise be attended with dyspnœa, is easy, because in this case there is no condensation of lung tissue. Nor should liquid effusions occasion any error, if it be borne in mind that the dulness caused by them is almost always bounded by horizontal lines. *Catarrhal pneumonia* is the most frequent cause of error. Here the course of the disease is very important, especially the change of symptoms, that is, the increase and decrease and altered character of the auscultatory signs as they occur in pneumonia and are absent in atelectasis. In pneumonia consonant râles and bronchial breathing are heard; in atelectasis, as a rule, only transmitted feeble vesicular breathing, with catarrhal sounds. Slight fever and pleuritic pains are accompani-

ments of pneumonia, whilst the retraction of the lower portions of the sternum, ribs, and intercostal spaces is absent.

TREATMENT.

Feeble infants, or children who have come into the world in a condition of very great exhaustion, should be made to cry, in order that air may be drawn into the lungs by the preceding deep inspiratory act. For this purpose they should be danced in the arms up and down through the air, the nasal mucous membrane should be tickled, the soles of the feet irritated with a brush, or the children taken into the fresh air, or sprinkled with cold water, even dipped for a moment into a cold bath, or a strong stream of cold water poured upon them when in a luke-warm bath. Moreover, the mouth should be cleared of any mucus or meconium, and thoroughly cleansed. Insufflation of air into the lungs through the child's mouth, a method formerly very highly prized, and still much in vogue, should, if practised, be attended with great care, lest vesicular and interlobular emphysema may be thereby produced.

Further, the children should always be warmly clothed, as the season of the year requires; they should be kept thoroughly dry, in order to avoid the danger of a catarrh from a wet diaper. A warm bath should be given daily, followed if necessary by a cold sponging. In case the children nurse badly, the mother's milk should be given with a tea-spoon. They should not be allowed to sleep too long at a time, and on being awakened the respiration should be stimulated by some irritation of the skin, such as brushing the soles of the feet.

In *collapse of the lung* attention should be given to the primary catarrh, and to the accumulations of mucus which may occur in the bronchi, for the removal of which expectorants, or, better still, emetics should be administered. The best form of giving the latter is Hufeland's emetic (ipecac., twenty-three grains; tartar emetic, from one-half to one grain; oxymel of squill, two and a half fluid drachms; water, ten fluid drachms; shake, one teaspoonful every quarter of an hour). Emetics have the further advantage that the deep and powerful inspiration

immediately preceding the act of vomiting has a directly curative effect upon the atelectasis. Rachitical children, or such as suffer from debilitating intestinal catarrh, should receive additional treatment. Especially should their strength be sustained by tonics, such as iron, quinine, and cod-liver oil. I have also in such cases had excellent results from the use of Trommer's malt extract, both the common variety and especially that which contains iron. Certain circumstances require the use of stimulants, of which the stronger wines should have the preference.

The patient's surroundings should be rendered as favorable as possible, for the injurious effects of bad air, and of a small, damp, and overcrowded dwelling upon the respiratory organs have been long recognized, and have lately been most thoroughly explained by Bartels. An atmosphere heavily charged with carbonic acid interferes with the elimination of that gas from the pulmonary air-cells. A highly carbonized blood then flows through the arterial system, and after slight stimulation the nervous system is thereby paralyzed, especially those portions governing the inspiratory muscles. A daily action of the bowels should be induced by the judicious use of mild cathartics, in order that the function of the diaphragm may not be impeded by over-distention of the abdomen. Any disorders of the circulatory system should be met by the use of stimulants.

In atelectasis from compression attention should be paid, first of all, to the primary disease and its symptoms, and also to any disturbances of the circulation. For compression of the lung, remaining after the absorption of a pleuritic exudation, a residence in sanitaria which have a moderate elevation should be recommended, such as Ischl and Reichenhall, and I have seen very excellent results from the use of compressed air, for which the last-named locality offers the best advantages.

ATROPHY AND HYPERTROPHY OF THE LUNGS.

Atrophy.—*Magendie*, Journ. de Physiol. exp., 1821, I., p. 78.—*Andral*, Précis d'anatomie pathologique. Paris, 1829.—*Hourmann* et *Dechambre*, Recherch. cliniq. pour servir à l'histoire des maladies des vieillards faites à la Salpetrière, Arch. gén de médic., Août, 1835, have given a most complete description of senile atrophy of the lungs, and the results of comparative examinations of the structure of the pulmonary tissue at the various ages of life.—*Prus*, Mém. de l'Académie de Méd. Paris, 1840, Tom. VIII., p. 1.—*Rampold*, Oesterr. Jahrbücher, 1845.—*Gairdner*, Month. Journ. of Med. 1851, May, July, Sept., and Canstatt's Jahrsber., III., p. 27.—*Durand-Fardel*, Trait. cliniq. et prat. des maladies des vieillards, 1854, p. 351. Translated into German. Würzburg, 1858, p. 425.—*Buhl*, Virch. Archiv, 1857, Bd. XI., p. 275.—*Geist*, Klinik der Greisenkrankh. Erlangen, 1857, p. 90 et seq.—*Rokitansky*, Lehrb. d. path. Anat., 1861, 3. Aufl., Bd. III., p. 47.—*Rindfleisch*, Path. Gewebelehre, 1. Aufl., p. 326.

Hypertrophy.—*Laënnec*, Traité de l'auscult., etc., Tom. I., p. 341.—*Andral*, Anat. Path., Tom. II., p. 514. Précis d'anatomie pathologique. Paris. 1829.—*Rokitansky*, Lehrb. d. path. Anat., Bd. III., p. 58. Compare also the bibliography upon Capillary Ectasis in the chapter on "Anæmia, Hyperæmia, and Œdema of the Lungs."

ATROPHY.

DEFINITION AND ETIOLOGY.

By atrophy of the lung we understand the wasting or consumption of the elastic and connective-tissue elements of the alveoli and pulmonary lobules, and also of the contractile muscular fibres, with simultaneous destruction of the capillary blood-vessels, in consequence of which the walls of the alveoli become very much wasted, perforated, or completely disappear. Atrophy of the lungs is a lesion of old age, called *atrophia*

senilis, also *emphysema senile*. It accompanies involution of other organs and a condition of general marasmus, and must therefore be considered as a result of imperfect nutrition. Very frequently atrophy of the lungs occurs at a comparatively early period, before a similar change has begun in other organs, or else it advances more rapidly than the involution of the latter.

Buhl describes an atrophy of the lungs, as observed by him in the autopsies of three cases of typhus fever, which was analogous to acute atrophy of the liver. It presented, however, evidences of an inflammatory process, and was unquestionably, as he himself admits, a high degree of desquamative pneumonia, in which the principal changes occur in the alveolar epithelium: a finely granular clouding going on to molecular destruction, with collapse of the pulmonary air-cells, and later a disappearance of the parenchyma by gangrene.

Pathological Anatomy.

On opening the thorax the lungs are seen as small organs, sometimes quite shrivelled up, occupying the posterior portion of the thorax, and, as a rule, they collapse but little. When only moderately atrophied they will still remain in contact with the side-walls of the chest, though their size is far less than that of healthy lungs; their anterior borders are thin and very much retracted, so that the pericardium and the trunks of the large cardiac vessels lie entirely uncovered. The lungs are small and diminished by one-third or more of their volume; moreover, they are of light weight, and feel soft, flabby, downy, or like an air-cushion. The lobes are sometimes adherent the one to the other. When cut into, the air escapes with a feeble, scarcely perceptible, soft crepitant sound. The atrophy is most marked in the superior lobes, especially the apices and anterior borders. It seems to originate in and extend from these points. Owing to the wasting of the pulmonary tissue in the anterior and superior portion, the posterior and inferior portion of the lung crowds upwards and forwards, causing a dislocation of the interlobular fissures, so that these from being normally horizontal become more vertical.

A section of the lung is dry, bloodless, and heavily loaded with pigment, which often gives it a uniformly black appearance. The tissue is relaxed, and of a coarse cellular structure, and whenever a number of adjoining air-cells have, by the wasting of their septa, coalesced, it resembles a network with large meshes. The coarser trabeculæ are composed, according to Rindfleisch, of the larger bronchial and vascular branches, as well as of some of the stronger interlobular septa.[1] If together with an extensive and diffused pigment-deposit, a high degree of œdema is present, the pulmonary tissue is often reduced to a black, decaying pulp. The deposit of pigment should not be looked upon as a result of rupture of blood-vessels, for microscopical examination shows that it is located partly within the obliterated capillaries and partly in their sheaths and in the walls of the bronchi, so that a diapedesis is the probable explanation.

The bronchi, especially the smaller ones, have thin, wasted walls, and are at the same time uniformly increased in diameter; very rarely do they bulge out irregularly. It is not uncommon to find, as a complication, a circumscribed or diffused bronchitis, with a scanty, viscid, or an abundant, purulent secretion, with consecutive atelectasis, which is often associated with small, indurated, deeply pigmented, lobular nodules. The bronchial glands are generally small, and thoroughly melanotic.

Notwithstanding the fact that many of the pulmonary capillaries are obliterated and destroyed, dilatation or hypertrophy of the right side of the heart is almost never met with in this disease, contrary to what is seen in true emphysema; the right half of the heart is, on the contrary, generally small, and often in a condition of so-called "brown atrophy." This is owing to the fact that an engorgement of the right side of the heart is avoided by a falling off in the quantity and quality of the blood coincident with the involution of the various organs of the body.

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[1] *Hourmann* and *Dechambre* distinguish three grades of pulmonary atrophy. In *the first*, the volume of the lungs is not diminished although the alveoli are enlarged and their walls wasted; in *the second*, by atrophy and wasting of the walls the air-cells communicate with one another, although still retaining their natural shape, which, however, is entirely lost in the *third grade*, when the walls of adjoining alveoli completely disappear, so that the latter coalesce and form irregular cavities.

Such an hypertrophy or dilatation of the heart can only occur in those cases in which the pulmonary atrophy considerably precedes the deterioration of other and particularly the hæmatopoëtic organs. The diaphragm is thin, flabby, shrivelled, and pale. The thoracic muscles are atrophied, and even the form of the thorax is materially changed.

Symptomatology.

The most prominent symptom, *dyspnœa*, is due to the diminution or entire loss of the elasticity and contractility of the pulmonary tissue, properties which are indispensable for the proper performance of the expiratory act; it is also partly caused by the reduction of the respiratory surface due to the obliteration and destruction of the capillaries. Inasmuch as the necessity of the respiratory act depends, according to Frey, upon the quantity of the blood, the dyspnœa will be greater in proportion as the atrophy of the lungs precedes, and is more pronounced than, the involution of the rest of the body. Further, the inflexibility of the thorax and atrophy of the respiratory muscles have their share in the production of dyspnœa.

Another equally important symptom is *cyanosis* of the cheeks, lips, hands, and feet, with which œdema of the extremities is not unfrequently associated. This cyanosis is due to the destruction of numerous capillaries, and to the severity and extent of the coincident catarrh.

Inspection generally reveals at once some anomaly in the shape of the thorax. The wasting of the cervical and dorsal intervertebral cartilages, which of course in each instance is slight, but in the aggregate exerts considerable influence upon the shape and position of the thorax, causes more or less kyphosis.

The principal effect of this kyphotic distortion is upon the ribs, exerting, according to Geist, a tension upon their vertebral articulations, the effect of which is to increase the posterior curve of the ribs on both sides of the thorax, whereby the sternum is sometimes thrust forwards after the manner of the so-called chicken-breast, and the thorax is flattened laterally. The same

distortion brings the ribs nearer one another, narrowing the intercostal spaces and shortening the long diameter of the thorax. The cavity of the chest is thus considerably diminished, and, according to Niemeyer, assumes a permanent position of expiration. Since the expansion and expansibility of the lungs are intimately dependent upon the capacity and mobility of the thorax, the influence of these thoracic distortions upon the atrophy of the pulmonary tissue in most cases should be taken into account. The whole thorax participates in the act of inspiration, and the expiratory subsidence follows quickly, so that Biermer describes the respiration as of a short and frequently sighing type.

Percussion over the lungs yields a strikingly clear and loud resonance, because the thoracic walls vibrate more easily and perfectly on account of the atrophied condition of the muscular covering and ribs. The extent, however, of the resonance is less than in health, by reason of the atrophy and diminished volume of the lungs, which allow the diaphragm and liver to extend from one to one and a half intercostal spaces further upwards. For the same reasons, the heart coming in contact with the thoracic parietes over a larger space, the extent of præcordial dulness is increased. The impulse of the heart can be distinctly seen and felt in its normal position, or from one to one and a half intercostal spaces higher upward.

Auscultation generally reveals, aside from râles due to a coincident catarrh, roughened, vesicular breathing, the roughness of which, according to Niemeyer, is in proportion to the difference between the calibre of the terminal bronchi and the dilated pulmonary air-cells.

Diagnosis.

The diagnosis is based upon the diminished volume of the lungs, the extension upwards of the diaphragm, the uncovered position of the heart, and the flattened thorax. These conditions serve to distinguish simple atrophy of the lungs from the ordinary vesicular emphysema, in common with which it presents the features of rigid thorax, dyspnœa, cyanosis, and occa-

sionally conditions of œdema and catarrh. Diminished volume of the lungs, with flattening of the chest, is also seen after absorption of an abundant pleuritic exudation; but in such a case the deformity is always unilateral, and can only take place in young persons whose thorax is still flexible.

TREATMENT.

Of course any treatment for a direct effect upon this disease is out of the question. It should be purely symptomatic, and confined to an amelioration or cure of the catarrh which augments the dyspnœa and cyanosis. Attention should also be paid to the maintenance of the patient's strength and condition of nutrition by means of good and easily digestible food, and by breathing warm, healthy, fresh country air. Of course the appearance of other symptoms should be met with appropriate treatment.

HYPERTROPHY.

Where a lung appears to be enlarged, one should be very guarded in assuming, from this fact alone, an hypertrophy of that organ, by which is understood an increase in the size of the organ, and a proliferation of the elements composing the lung tissue. The enlargement is generally due to filling of the air-cells with serous fluid (œdema) or with exudative material, or else we find an inflated condition of the alveoli, or even an atrophy of the lung tissue with distention of the alveoli, a lesion which we will treat of elsewhere as "Pulmonary Emphysema."

Laënnec, relying upon the statement of Morgagni, that with emphysema of one lung the healthy one increases in size, mentions a vicarious hypertrophy of the lung which takes place whenever the other one is destroyed or incapacitated, as, for instance, by pneumonia, hydrothorax, or by contraction of the thorax after pleuritic effusions or large pulmonary cavities. He states that this hypertrophy consists of a distention of the alveoli, with thickening and increased volume and elasticity of their walls, causing the whole lung to be larger and denser. Therefore, upon the removal of the sternum, in many cases the

lung bulges forward out of the thoracic cavity, which has become too small for it. Such a pathological condition, according to Laënnec, frequently arises in a very short time. Frey believes that an hypertrophy of the lung tissue takes place whenever the increased extent of the respiratory motions is associated with an augmented nutrition of the lung cells which have been moderately distended thereby; but that pulmonary emphysema occurs whenever there is distention of the lung-cells with disordered nutrition.

We more than doubt the existence of such an hypertrophy, and unhesitatingly class it with ordinary vesicular emphysema. The alleged hypertrophy with proliferation of the alveolar wall tissue is only apparent, and is due to compression of the healthy alveoli by the dilated pulmonary lobules, as will be further explained when treating of emphysema.

A *second* form of hypertrophy of the interlobular connective tissue, described by Rokitansky, has an inflammatory origin, and belongs to the category of interstitial or cirrhotic pneumonia.

A *third* form consists, according to Rokitansky, in an increased development of the connective-tissue framework of the air-cells, causing the parenchyma to appear dense, turgid, and very resistant. Upon section the alveolar walls are thick, do not collapse, and resemble a coarse network. This pathological condition, which is especially apt to be associated with mitral disease, is described by Scoda also as an hypertrophy due to irritation, and corresponds with Hasse's *brown induration* and with Virchow's *brown* or *pigment induration*. Very careful recent investigations have given rise to some doubt as to whether this is a *real hypertrophy*, or at least tend to show that any proliferation of interlobular connective tissue is incidental—a point which has already been discussed in detail in the chapter upon hyperæmia of the lungs.

We come, therefore, to the conclusion that hypertrophy of the lungs does not exist, and that what has hitherto been considered as such is really some other pathological condition.

PULMONARY EMPHYSEMA.

Laënnec, Traité de l'auscultation, 1819, I., 348.—*Magendie*, Journ. de. physiologie, T. I., 1825.—*Andral*, Anatomie pathol., T. II., 1829, 524.—*Piédagnel*, Recherch. anat., etc., Journ. de Phys., T. II.—*Bouillaud*, Dict. en XV. vol., Vol. VII., p. 121. Paris, 1831.—*Townsend*, Encyclop., Vol. II., 78, Art. Emphysema.—*Lambert*, Cons. gén. sur l'emphys. Paris, 1834.—*Ymly*, Sur l'emphys. pulm. Paris, 1834.—*Louis*, Mém. de la soc. d'observ., 1836, T. I., p. 160, and Dict. de méd., T. XI.—*Stokes*, Dublin Journ. of Med. Science, Vol. IX., Nos. 25 and 26, 1836, and Diseases of the Lungs, 1837, Behrend's translation, p. 96.—*Lombard*, Mém. de la. soc. de phys. et d'hist. nat. de Genève, 1837.—*Woillez*, Recherches. Paris, 1838.—*Budd*, The Lancet, Dec. 31, 1839.—*Nienbaur*, De emphysem. pulm. Groningen, 1839.—*Monneret* et *Fleury*, Compend. de méd. prat., T. III., p. 193. Paris, 1839.—*Beau*, Arch. gén., 3me sect., T. IX., p. 377. 1840.—*Roger-Collard*, Bull. de l'acad. de méd., 1842, T. VIII., p. 710.—*Budd*, Lond. Med. Gaz., Vol. XXV., 1842, p. 490.—*Adelon*, Bull. de l'acad., T. VIII., p. 686; Gaz. Méd., 1843, p. 859.—*Prus*, Mém. de l'acad. de méd., Vol. X., p. 655. Paris, 1843.—*Gavarret*, De l'emphys. pulm. Thèse. Paris, 1843.—*Chomel*, Gaz. des hôp., 1844, No. 57.—*Lebert*, Physiol. pathol. Paris, 1845, I., p. 203.—*Rampold*, Oesterr. Jahrbücher, 1845, I., 6.—*Fueter*, Schweizer Cant. Zeitung, 1845, I., 4.—*Fuchs*, Abhandlung über Emphysem. Leipzig, 1845.—*Mendelssohn*, Der Mechanismus der Respirat. und Circulation. Berlin, 1845.—*Wunderlich*, Handb. der Pathologie und Therapie. Stuttgart, 1846, III.—*Strohl*, Gaz. méd. de Paris, 1848, Nos. 33, 36, 40, and 42.—*Rainer*, Med.-Chir. Trans., Vol. XXXI., 1848.—*Rosignol*, Recherches. Brux., 1849.—*Sibson*, Lond. Med. Gaz., Jan., 1849.—*Donders*, Nederl. Lancet, Mai, Juli, Oct., 1849, Oct., 1852.—Zeitschr. f. ration. Med., N. F., Bd. III., 1 and 3, 1853.—*Gairdner*, Ed. Month. Journ., Vols. XII. and XIII., 1851.—*Frey*, Archiv für physiologische Heilkunde, 1851, Bd. X., p. 44.—*Dittrich*, Prag. Vierteljahrschr., Jahrg. VIII., Bd. 3, 1851.—*Wachsmuth*, Zeitschr. f. ration. Med., IV., p. 212.—*Rilliet* et *Barthez*, Traité de mal. Paris, 1853, T. I.—*Guillot*, Arch. gén., 1853, Août.—*Gallard*, Arch. gén., 1854, 5me sect., T. IV.—*Ozanam*, Arch. gén., 1854, Jan.—*Cohn*, Günsburg's Zeitschr., 1854, Bd. V.—*Baerent*, De telæ, etc. Dorpat, 1855.—*Barton*, Dublin Hosp. Gaz., No. 6, 1857.—*Jenner*, Med.-Chir. Trans., Vol. XL., 1857; Med. Times and Gazette, Jan., 1857; Brit. Med. Jour., Nos. 4 and 5, 1857.—*Bermont*, L'emphys. Paris, 1858.—*Ziemssen*, Deutsche Klinik,

1858, No. 16.—*Hewitt*, On Vesicular Emphysema. Liverpool, 1858.—*Freund*, Der Zusammenhang zwischen Lungenkrankheiten, etc. Erlangen, 1859.—Verhandlungen der med. phys. Gesellschaft zu Würzburg, 1859, Bd. IX., p. 223.—*Waters*, Boston Med. and Surg. Jour., July 28, 1860; Lancet, 1860.—*Virchow*, Deutsche Klinik, 1860, 47.—*Gerhardt*, Der Stand des Diaphragma's. Tübingen, 1860.—*Scoda*, Allg. Wien. med. Zeitung, 1861, Nos. 11, 45, 46, and 47.—*Hervieux*, Arch. gén. de méd., 1861.—*Royer*, Journ. f. Kinderkr. Wien, 1862, Heft 11 and 12.—*Niemeyer*, Berl. med. Wochenschr., 1864, Nos. 44–46.—*Schmidtlein*, Deutsche Klinik, 1864, Nos. 20, 22, 27, and 28.—*Oppolzer's* Vorlesungen über spec. Pathol. u. Therap., 1866, p. 520.—*Villemin*, Arch. gén., 1866, Oct. and Nov.—*Biermer*, in Virchow's Handbuch der spec. Pathol. u. Therapie, Bd. V., Abth. 1, Liefg. 5.—*Headlam-Greenhow*, Lancet, 1867, No. 23 et seq.—Clinical Lecture, The Lancet, May 9 and June 13, 1868.—*Waters*, Diseases of the Chest. London, 1868.—*Johnson*, Brit. Med. Jour., June 27, 1868.—*Bayer*, Archiv f. Heilkunde, 1870, p. 360.—*Hauke*, Ein Apparat zur künstl. Respiration. Wien, 1870.—The same, Allg. Wien. med. Zeitung, 1870, 34, and Wochenbl. der Wien. Aerzte, 1870, No. 30.—Nachtrag zur Brochüre. Wien, 1872.—*Waldenburg*, Berl. klin. Wochenschr., 1871, 45; 1873, Nos. 39, 40, 46, and 47.—*Berkard*, Lancet, No. 25, 1871.—*Isaaksohn*, Virchow's Archiv, 1871, Bd. 53, p. 466.—*Rohden*, Wien. med. Wochenschr., 1873, Nos. 17 and 18.—*Gerhardt*, Berl. klin. Wochenschr., 1873, No. 3.—*Riegel*, Die Athembewegungen. Würzburg, 1873, p. 95.—*Eichhorst*, Deutsch. Arch. f. klin. Med., 1873, Bd. XI., Heft 3.—*Lebert*, Klinik der Brustkrankheiten. Tübingen, 1874, Bd. I., p. 324.—*v. Liebig*, Bayer. ärztl. Intelligenzblatt, 1874, I. ser., No. 4.—*Sommerbrodt*, Berl. klin. Wochenschr., 1874, Nos. 15 and 31.—*Niemeyer*, Handbuch der Path. u. Ther. Berlin. Compare also the hand-books of pathological anatomy by *Hope*, *Hasse*, *Förster*, *Lebert*, *Rokitansky*, *Rindfleisch*, and others.

HISTORY.

It appears from certain writings of Bonnet and Morgagni that emphysema was well known to the older physicians, although for the most part they referred the symptoms to hydrothorax. Laënnec was the first to give an accurate description of the structural changes, and his portrayal of the symptoms was so precise that it still serves as the standard, to which there is but little of importance to be added. Whatever it may have lacked in precision has been supplied by Louis, Lombard, and Rokitansky, in regard to the pathological anatomy of the disease, and by Scoda in regard to its symptomatology and diagnosis.

The view put forth by Laënnec, that emphysema is a simple enlargement of the pulmonary alveoli, has been contested in various quarters. Thus, Andral looked upon it as a condition consisting of both hypertrophy and atrophy, whilst Louis described the changes in the lungs as hypertrophic, and others again as atrophic. Rokitansky first presented the disease in its true light, when he recognized that the dilatation of the pulmonary alveoli was coupled with atrophy of the lung tissue. Laënnec had previously referred the disease to a mechanical origin, namely, increased pressure of air within the alveoli during inspiration—a negative inspiratory pressure. Jenner, Mendelssohn, and others attach greater importance to the heightened positive pressure of expiration, and still others admit a primary tissue change in the lungs.

The measures which were employed in the treatment of the fully developed disease were of little avail. It is only in recent years that an effectual remedy seems to have been found in the inhalation of compressed and rarefied air, by means of the portable apparatuses of Hauke and Waldenburg.

DEFINITION.

By the term *emphysema* (ἐμφυσημα, from ἐν and φυσάω, or φυσέω, I blow) we understand the inflation or infiltration of the interstitial connective tissue of an organ with air. The expression *pulmonary emphysema* was first introduced by Laënnec, and it denotes two pathological conditions, which differ from each other in their locality. One of these—called *vesicular* or *alveolar emphysema*—consists of an enlargement of the pulmonary vesicles or lobules, due to dilatation (stretching of the lung, pneumonectasis, or alveolar ectasia), atrophy and wasting (rarefaction [Rokitansky]), or rupture of the alveolar walls. The other—*interlobular emphysema*—consists in an accumulation of air in the interlobular and subpleural connective tissue.

Vesicular emphysema (the pulmonary emphysema *par excellence*) either occurs as an idiopathic disease, *i.e.*, as *substantive* or *substantial* emphysema, or it is developed in connection with other affections of the pulmonary parenchyma, in which

a certain number of alveoli have become impermeable to the inspired current of air, so that their function has to be undertaken by the other alveoli, which thereby become immoderately distended, constituting *vicarious emphysema*. In speaking of emphysema, the first variety, the substantive, is always understood, unless otherwise specified.

ETIOLOGY.

Pulmonary emphysema is unquestionably one of the commonest diseases, easily recognized during life when fully developed, but in its slighter forms discoverable only after death. It most rarely occurs as interlobular emphysema, and is then probably always complicated with the alveolar variety. The latter, in its substantive form, is often associated with bronchial catarrhs and with certain occupations which require forced inspiratory and expiratory acts. In its vicarious form it attends most of the diseases of the pulmonary parenchyma and certain changes within and in the neighborhood of the chest. Statistics in regard to its relative frequency furnish few data of positive value, since many of those who are affected with emphysema do not resort to hospitals or even seek medical advice, whilst the majority of cases of vicarious emphysema are recorded under the head of some other disease. And yet it is unmistakable that emphysema is oftener met with in winter, and in the more inclement seasons of the year, in cold, damp, and unsheltered localities, where pulmonary affections prevail, than under opposite circumstances. Moreover, the statistics of autopsies may be expected to show a larger percentage of cases than would appear from clinical observations, since the lighter, unrecognizable cases usually escape the latter.

Among the *predisposing causes* of pulmonary emphysema is to be reckoned *heredity*, as was first pointed out by Jackson and Louis. Many observers attach great importance to it, attributing half the cases to its influence. Thus, Jackson believes he found an hereditary predisposition in twenty-eight out of fifty persons affected with emphysema. A *congenital* emphysema, such as is assumed by many, does not exist, since

a lung to which air has never had access can scarcely be emphysematous.

The male *sex* is more frequently affected than the female—not so much on account of any difference in the lungs of the two sexes as because of the external relations, the occupations, and the mode of life peculiar to men. Emphysema is seldom met with in children and young persons, but occurs more commonly in adults, and increases in frequency as age advances. The reason of this lies in the frequency and the more protracted course of chronic bronchial catarrhs, as well as in certain structural changes in the pulmonary parenchyma incident to the decline of life.

Of 858 cases observed by Lebert, in sixteen years of his practice, 16 per cent. occurred between 20 and 30 years of age, 28 per cent. between 30 and 40 years, 40 per cent. between 40 and 60 years, 11 per cent. between 60 and 70 years, and 3.7 per cent. between 70 and 80 years. But, since the beginning of the disease should be set down as occurring, on an average, at least five years prior to its coming under observation, and not infrequently as much as ten years, we should allow, he thinks, that in more than two-fifths of the cases the disease is developed during youth and the prime of life.

Vocation and the *mode of life* may have some influence, as favoring the occurrence of catarrh, or as involving a certain strain on the respiratory function, as in porters, musicians, etc. A circumstance of far greater importance in predisposing to the development of emphysema than those thus far mentioned, consists in a *nutritive* disturbance of the lung tissue, which we shall consider more particularly hereafter.

The *exciting causes* of *vicarious emphysema*, which may be either acute or chronic, are: (1), morbid changes within or adjoining the thorax, which, by the pressure which they exert from without upon the lungs, render a greater or lesser portion of the latter impermeable to air. This may happen in consequence of pleuritic and pericarditic exudations, hydrothorax and hydropericardium, intrathoracic and mediastinal tumors, aortic aneurisms, goître, curvatures and tumors of the spine, etc.; (2), obstruction of the air-passages by foreign bodies, impaction of the air-cells and bronchioles with solid or fluid material, as in hypostasis, catarrhal and croupous pneumonias, and in obstruc-

tion of the smaller bronchi by mucous or croupy masses, with consequent collapse of the pulmonary parenchyma ; (3), chronic inflammatory *interstitial proliferation of connective tissue*, with secondary shrinking and wasting of the parenchyma, also *new-formations*, such as carcinoma, tubercle, etc., with consequent circumscribed atrophy of lung tissue ; (4), partial, tense *pleuritic adhesions*, as was first brought to general notice by Donders.

In ordinary deep inspiration the more movable portions of the thorax, the anterior wall and the diaphragm, produce such a decided enlargement of the thoracic cavity that the normal inspiratory inflation of the subjacent portions of lung is not sufficient to fill out the void. On the other hand, the summit of the chest and that portion which borders on the spinal column are only very slightly enlarged, and thus only a very limited space is utilized for the expansion of the corresponding parts of the lungs. To compensate for this disproportion, the lung shifts its position from behind forwards, and from above downwards, with each inspiration, and *vice versâ* with each expiration. If, however, there be firm and extensive adhesions between the two layers of the pleura, this movement cannot take place, and, consequently, the posterior portions of the lung take on a condition of atelectasis, whilst the anterior and inferior portions become affected with vicarious emphysema.

We apprehend that this is true only of pleural adhesions which are of considerable extent ; but that mere ribbon-like adhesions, contrary to the opinion of Donders, are not sufficient for the production of emphysema. In such cases, as Biermer suggests, we must infer the coexistence of other causes, such as bronchial catarrh and strained respiration.

Substantive emphysema may be developed (1) in all diseases which are accompanied by *severe and protracted paroxysms of coughing*, such as ordinary bronchitis, particularly the chronic, dry form which affects the smaller bronchi,[1] whooping-cough, chronic bronchial croup, and, although more rarely, hysterical cough, and the severe coughing which follows the inhalation of irritating gases ; (2) in *any other forced movement of the breath*,

[1] According to *Traube*, this is the only form of bronchitis which leads to emphysema ; diffuse bronchial catarrh, with homogeneous, muco-purulent, and nummular sputa, with coarse râles at the posterior and lower portions of the chest, no matter how excessive may be the dyspnœa, never gives rise to dilatation of the lungs.

such as occurs in certain occupations—playing upon wind instruments, singing, the use of the blowpipe, etc.—also in the bearing-down efforts attending parturition or obstructed defecation, as well as in lifting heavy weights. These acts involve deep inspirations and expirations—the latter being chiefly effected by powerful action of the abdominal muscles, and generally accompanied by impeded egress of the air contained in the chest through the larynx, the mouth, and the nose.

According to Rokitansky, emphysema of the upper lobes of the lungs is developed in persons who are otherwise healthy, but who pursue their avocation in a sitting, stooping posture, especially if at the same time they make laborious use of their upper limbs. The stooping posture, by compressing the abdomen, leads to a suspension of the action of the diaphragm, which is compensated for to a certain extent by the action of the other great respiratory muscles of the upper part of the thorax. The increased expansion thus produced in the upper portion of the chest, and shown in the slower and therefore deeper inspiration, is, according to Rokitansky, the cause of emphysema.

In the dead body we often meet with dilatation of the air-cells near the anterior surface of the lungs, developed apparently during the agony. It is the result of the high degree of dyspnœa which attends death by suffocation, such as occurs in acute œdema of the lungs and hæmoptysis; or where there is a deficiency of blood in the pulmonary arteries, with consequent diminished interchange of gases, as happens in cholera and in embolism of a large branch of the pulmonary artery. This dilatation depends upon a disproportion between the inspiration and the expiration, since in forced inspiration, involving as it does a rapid and unnatural expansion of the chest, the alveoli of the anterior portions of the lungs are uniformly and excessively dilated, while the forces which serve for expiration are, from the occurrence of paralysis, no longer adequate to the expulsion of the air. This condition, which Kluge designated as *insufflation*, may be recognized even during life by the increased area of the lungs and by the heart being overlapped by lung tissue. It is met with in the bronchitis of children and in the bronchial asthma of adults.

In the former instance the puffing up of the lung is due to the fact that inspiration overcomes the narrowing of the smaller

bronchi due to the bronchitis, and forces the air into the alveoli, whilst expiration, on the other hand, the strength of which generally surpasses that of inspiration, exercises, according to Niemeyer, a uniform pressure upon the alveoli and the contracted bronchioles, by means of the energetic contraction of the abdominal muscles and the upward impulsion of the diaphragm. The pressure upon the bronchioles impedes the egress of air from the alveoli. In *asthma*, according to Biermer, there is a tonic spasm of the muscles of the medium-sized and finer bronchial ramifications. The impediment thus offered to the ingress of air is sufficiently overcome by the inspiratory effort; but expiration produces the same simultaneous pressure upon the alveoli and upon the bronchi, already narrowed by the spasm, so that the egress of air is impossible so long as the spasm lasts.

Puffy distention of the air-cells differs, however, from true emphysema in this, that it lacks the anatomical condition of atrophy and wasting of tissue, which will presently be discussed. It is possible, nevertheless, that a true emphysema may originate in mere distention, provided the inspiratory stretching of the alveolar walls be sufficiently protracted and frequently enough repeated to cause at last real atrophy.[1]

Interlobular and *subpleural* emphysema, which is of relatively rare occurrence, is formed by rupture of the alveolar and infundibular walls, and the escape of air into the interlobular and subpleural connective tissue. It is the result of an inordinately high atmospheric pressure within the air-cells. This would appear to take place, although rarely, in a sort of vicarious manner, in case of the failure of a considerable number of the alveoli to expand during the inspiratory inflation of the chest (Niemeyer). There can be no theoretical objections to the possibility of this inspiratory mode of development, but it is doubtful if it occurs in practice. When it exists, we must certainly attach chief importance to the expiratory pressure and to certain lesions of nutrition in the pulmonary parenchyma. It is most frequently occasioned by the severe paroxysms of cough

[1] *Walshe* relates a case of emphysema in a child which died two hours after its birth. In this instance the emphysema was due to severe dyspnœa, of cerebral origin, without any bronchitis, pneumonia, or atelectasis.

which occur in croup, whooping-cough, and dry bronchitis, particularly during childhood, when the alveolar tissue, which, although very elastic, is tender withal, gives way under the high direct pressure of expiration. It may arise also from the frequent paroxysms of coughing which follow the inhalation of irritating vapors (Guéneau de Mussy's case, cited by Depaul[1]). Furthermore, it may be caused by forced inspiration and expiration in cases of excessive dyspnœa and impending asphyxia, and, according to Leroy d'Étiolles, by insufflation of air into the lungs of children born apparently dead.

THEORY OF THE DEVELOPMENT OF EMPHYSEMA.

Knowing as we do, in a general way, the causes of pulmonary emphysema, opinions are still divided as to the mode and manner of its development. Some explain the changes in the lung as due to the purely mechanical action of the air contained within the alveoli, under the influence of an increase either in the so-called negative inspiratory pressure or in the positive expiratory pressure. Others assume the existence of a lesion of nutrition in the tissue of the alveolar walls. In our opinion both are right, since neither one of these three explanations can be held as solely and exclusively sufficient in every case of emphysema. One or another of them may be of the more importance in a given case, or one of them may figure alone, while the others recede wholly from view.

According to Laënnec, the existence of bronchitis impedes the entrance of air into the alveoli by causing a swollen condition of the mucous membrane of the smaller bronchi, with accumulation of secretion in them. This impediment, however, may be overcome by deep and forcible inspiration, whereas the expiratory force is not sufficient to expel the imprisoned air. Thus the alveoli become highly distended, and the distention is still further increased by the fact that the contained air, which is closely shut in by the secretion, is expanded by the heat of the body. This combined inspiration and expiration theory may have its value in certain cases of dilatation of the pulmonary

[1] Gaz. méd. de Paris, 1842.

parenchyma, as met with in the bodies of those who have died with marked dyspnœa, but yet it does not suffice to explain emphysema.

Whether the air expands within the lungs, and, if so, to what extent, is unknown; it can indeed be only trifling in the case of that contained within the alveoli, since it must already have been warmed in its passage thither. Moreover, Louis has pointed out that the emphysema is found not only in those alveoli which belong to the affected bronchi, but also in portions of the lung which show no bronchial lesions.

In this connection we come naturally to speak of Williams' theory, according to which *vicarious* emphysema is developed in certain affections of the lungs, in which a portion of the organ, of greater or less extent, becomes impenetrable to air, on which account the negative inspiratory pressure gives rise to emphysema in the alveoli corresponding to those bronchi which are not clogged. This view is received by many, and acknowledged to be correct. But some, not satisfied with this, attribute the development even of substantive emphysema to the agency of the heightened negative inspiratory pressure. That the pulmonary alveoli become distended by means of an exalted inspiratory pressure, coupled with expiratory forces relatively weak in expulsive action, and that emphysema may be developed in consequence of atrophy of the tissue, resulting from the long continuance of this condition (the *permanent inspiratory expansion* of von Niemeyer), we must concede on theoretical grounds. But, that in lungs which are in other respects healthy, where the expiratory forces are not weakened, and where, therefore, the egress of air is unimpeded, emphysema may be developed by the mere increased force of inspiration alone, is not yet in any way sanctioned by experience. It is to be presumed, moreover, that, with a normal condition of the lungs and an equable distribution of air to all the alveoli, a heightened air-pressure will not produce an excessive and damaging distention of their walls.

In *vicarious emphysema* there is a diseased condition of the pulmonary parenchyma, by virtue of which the relations of its various parts undoubtedly undergo a change. It then happens

that the air, even in the normal inspiratory act, is unequally distributed, and that, if a number of the pulmonary lobules be impermeable, a corresponding compensatory increase of pressure is brought to bear upon those portions of the lung which are still pervious. To what extent, however, this increase of pressure must be carried, in order to occasion not only a temporary distention, but also a concomitant atrophy and wasting of the tissue, is not to be determined either in general or in particular instances. Even were we able to fix upon a definite degree of increase of pressure as the limit of that which the elasticity of the lung substance would enable it to withstand, it would still always be fair to suppose that the air would be apportioned in equal quantity to *all* the alveoli, and that the walls of each one of them would possess an equal power of resistance. Yet both of these conditions are wanting in vicarious emphysema, since even those portions of the lung which have thus far escaped the original disease cannot be looked upon as perfectly normal lung tissue; hyperæmia, œdema, etc., cannot but exercise an influence upon the resisting power of the walls, a point which we shall specially consider further on.

Now, on the other hand, let us not underrate a circumstance which has been adduced by Biermer, namely, that a certain number of these patients breathe only superficially, whether it be that they have a diminished *besoin de respirer*, or that their inspiratory muscles are atrophied, or that their costal cartilages are ossified, or that pain causes them to restrain their breathing. It is not improbable that in such cases the negative inspiratory pressure is less than normal, and that therefore the above-described effect upon the alveoli is not exerted at any time. We cannot, then, as many do, admit the inspiratory pressure as the sole cause of vicarious emphysema in the majority of cases, for it is unquestionable that lesions of nutrition, or even an increase of expiratory pressure, with its accompanying paroxysms of cough, frequently exert an influence. On the other hand, however, we will not wholly deny the effect of inspiratory pressure, as indeed it is of common occurrence.

It is to be admitted (1) in cases in which a concomitant positive expiratory pressure is either altogether wanting, or, from

the existing conditions, can be credited with only a slight effect; (2) in cases of vicarious emphysema in which the inspiratory act is of normal or increased force; (3) when the emphysema affects those parts of the lung which undergo the greater pressure from the thoracic walls in active expiration (since, in reality, the muscles which are called into action in forced expiration, as well as the counter-pressure on the part of the thoracic wall, strengthen the resistance which the walls of the pulmonary alveoli offer to the positive air-pressure); (4) in the majority of the cases in which the permanent inspiratory form of thorax is found, which remains to be described.

In diseases attended with cough and followed by emphysema, the shock of the cough, or, in other words, the exaggerated expiration, long ago attracted notice, it is true; but less importance was attached to it than to the deep inspiration which follows forced expiration (Rokitansky). In 1845 Fuchs pointed out that, by means of forced expirations with the glottis narrowed or closed, the air within the chest was subjected to great pressure, which affected the alveolar walls on all sides alike and distended them inordinately, unless a sufficient counter-pressure from without were afforded by the bony thorax or by the contracted muscles. Mendelssohn fortified this *expiration theory* by showing, that in the act of expiration the lung is compressed from below by the ascent of the diaphragm and by the contraction of muscles which are pre-eminently efficient by reason of their situation at the lower portion of the thorax, thus producing an expiratory current upwards to the trachea. This current, as Mendelssohn believes, interferes with the egress of air from above, *i.e.*, from the upper lobes, heightens the pressure in that situation, and thus distends the alveoli of the upper portion of the lung. Jenner also upholds this doctrine by showing that in expiration the lungs do not undergo an equal pressure in all their parts, and that emphysema occurs in just those portions upon which the thoracic walls and the contracting muscles exercise a minimum of counter-pressure from without. These portions are the apex and the root of the lung. Von Ziemssen, too, has been enabled to corroborate this view by an interesting case.

He observed in an athletic man, forty-five years of age, a congenital deficiency of the pectoralis minor muscle and of the entire sterno-costal portion of the pectoralis major, so that the muscles of the four upper intercostal spaces were covered with nothing but skin. On quiet inspiration a considerable sinking in was noticed in these situations, which was succeeded, on quiet expiration, by a return of fulness. On forced expiration, however, the soft walls of the four upper intercostal spaces were puffed out to the extent of a line or a line and a half beyond the level of the ribs. On producing contraction of the muscles of one of these intercostal spaces by means of the faradic current, coughing was no longer attended with bulging of that particular intercostal space, although the latter continued to occur in the others.

Von Ziemssen accordingly explains the production of emphysema in the following manner: During quiet expiration an escape of air occurs into the bronchi from all the lobes alike, without muscular contraction, and due solely to the contractility of the lung, the weight of the thoracic wall, and the elasticity of the intestinal gases and the abdominal muscles, whilst, on the other hand, in forced expiration, the auxiliary expiratory muscles, which, however, do not extend above the fifth rib, are also brought into action. In case the glottis is closed, the air within the chest is thus subjected to an increased pressure (of 57 mm. Hg.), which bears upon the alveolar walls equally in all quarters. It is only in those situations in which the auxiliary expiratory muscles exercise a counter-pressure that the alveoli are not distended to an unnatural degree; but where, as in the upper portions of the thorax, this support is lacking, the alveoli are distended, and a frequent repetition of this occurrence may give rise to the changes which are characteristic of emphysema.

The situation of emphysema in the upper lobes of the lungs is, in most instances, particularly suggestive of the etiological agency of heightened positive expiratory pressure. Even in ordinary breathing, and particularly on forced inspiration, there is a sinking in of the supraclavicular region, which rises again on the occurrence of ordinary expiration; but, on forced expiration, as in coughing, it is bulged out, as may easily be ascertained by palpation. But if, as Rokitansky maintains, emphysema is due not to the expiratory pressure in coughing, but to the deep inspiration which occurs between the paroxysms of cough, the converse phenomenon should be apparent. It is

plain that in forced expiration a compression of the lower portions of the lungs occurs, whereby the contained air is subjected to a heightened pressure. This is particularly injurious to those portions of the lung which, like the apex, are destitute of the support afforded by the thoracic walls and by the contracted expiratory muscles. This pressure is intensified so soon as the egress of air is interfered with by narrowing or closure of the glottis. The air which is compressed within the lower lobes crowds upwards, and, instead of finding an exit through the larynx, gains entrance into the principal bronchus of the upper lobe, which is directed obliquely downwards.

There is another circumstance which we must not overlook, and which we have already mentioned as among the predisposing causes. I refer to certain *lesions of nutrition* in the pulmonary parenchyma, which doubtless enhance in no inconsiderable degree the effect of the increased inspiratory and expiratory pressure, and may indeed, even if there be no positive proof of the fact, serve as the occasion of the development of emphysema without any abnormality of inspiration or expiration whatsoever. Although we neither know the precise structural changes, nor are able to refer them with certainty to their causes, we cannot on that account ignore their existence or their significance in connection with emphysema. The admission of a lesion of nutrition is called for on the following grounds:

Louis formerly showed that the bronchial catarrh did not always precede the emphysema, but that, on the contrary, particularly in children, it did not make its appearance until after the emphysema had become developed. Certain textural lesions of the pulmonary parenchyma must therefore have been present, which, with the inspiratory and expiratory pressure either normal or moderately heightened, admitted of a distention and atrophy of the alveoli. In this light, indeed, must we look upon the so-called hereditary emphysema, in which it is not the emphysema itself that is inherited, but only the highly yielding and unresisting condition of the tissue. We thus find, although rarely, that all the members of a given family, of which I myself know an instance, will become affected with more or less pronounced emphysema on the occurrence of comparatively trifling

disturbances. Furthermore, emphysema may, although this likewise is uncommon, become developed without its cause being in any way discoverable, and the same etiological conditions may in one case produce a tolerably well-marked emphysema, whilst in another case they do not occasion a trace of it. Thus, only a few of those who play on wind instruments, and not by any means all children with whooping-cough, even if they have had the severest paroxysms, become affected with emphysema. Moreover, it scarcely ever happens that both lungs are emphysematous to an equal degree. Individuals may also, without prejudice to their lungs, expose themselves for a long time to conditions which are known to lead to emphysema. But these same conditions may, however, on the supervention of some disease of the pulmonary parenchyma, rapidly cause emphysema. A case observed by myself may here be mentioned:

A powerful and well-built regimental cornet-player, about thirty years old, who had before been perfectly healthy, who had always been able to blow vigorously and thus maintain a prolonged note, and whose playing was not embarrassed even by marching, fell ill with a double croupous pneumonia, which, however, passed into the stage of resolution on the seventh day. The patient afterwards felt well, and soon regained his appetite perfectly, and no cough remained. It was not long before he began to play again, practising industriously in his quarters without any special difficulty. About seven months subsequently this musician came to me, complaining that he could not draw so deep a breath as formerly in the interval between blasts, and that his wind was not sufficient for the maintenance of a prolonged note; he also got out of breath on walking, and could not play at all while marching. Physical examination showed considerable emphysema, from which he had not previously suffered. The supposition was obvious that the pneumonia, which had occurred seven months before, had left a structural change in the lung tissue, in consequence of which the parenchyma was unable to offer the necessary resistance to the very severe positive expiratory pressure occasioned by blowing.

The recognition of a disturbance of nutrition is further called for by the fact that pulmonary emphysema increases in frequency with the advance of age. This may be partly due to chronic bronchial catarrh, but, even in the absence of this, emphysema may be developed in consequence, indeed, of a senile physiological change. The rare occurrence of emphysema as the result of nervous and hysterical convulsive coughs, in spite of the frequently protracted course of the disease and the

certainly not inconsiderable positive expiratory pressure, shows, too, that some cause other than the mechanical one must contribute to the production of pulmonary emphysema. This form of cough occurs, for the most part, in young women with healthy respiratory organs, in whom the nutritive lesions in question are wanting.

These considerations suffice, I believe, to place textural disease of the lung in its true light in relation with the causation of emphysema, and to warrant its being included at least amongst the predisposing causes.

Another theory, brought forward by Freund, rests upon the idea of a nutritive lesion, situated primarily in the costal cartilages.

Somewhat short of the twentieth year of age, according to Freund, the ribs assume, from their central axis outwards, a dirty yellow color, and become softened, stratified, and coarse-fibred. As these fibres now have a tendency to bend and twist, roundish spaces and breaches form between them. The cartilage is thus pressed apart in its outer layers, and increases in volume in all directions. The tissue loses its flexibility and elasticity, becoming stiff and brittle. On account of this wayward growth, the two bony points, the one upon the rib and the other upon the sternum, between which the cartilage is inserted, are spread further apart, and thus the ribs are forced outwards and upwards, and the sternum forwards and somewhat downwards. By a further increase, the cartilage undergoes an exaggeration of its natural outward convexity, and accommodates itself to the level of the anterior extremities of the bony ribs, so that the thorax, as regards the six lower true ribs, loses its normal expiratory attitude, and assumes permanently that of inspiration. The permanent distention and enlargement of the thorax thus produced may lead to secondary distention of the lungs, and finally to emphysema.

However plausible this theory of *primary fixed dilatation of the thorax* may appear, we are nevertheless of the opinion that only a subordinate importance should be attributed to it. In case of the actual occurrence of the primary changes in the cartilage, as specified by Freund, and of which I have not yet been able to convince myself, it may well be supposed, as Niemeyer has pointed out, that the selfsame causes, such as forced expiration or inspiration, may give rise to pulmonary emphysema and at the same time to the lesion of the cartilage. Both induce an inordinate stretching and tension of the cartilage, and perhaps also an irritative condition which may lead to struc-

tural changes. It is unmistakable, too, that stases of blood in the intercostal and mammary veins, in consequence of deficient emptying of the right cavities of the heart during forced expiratory efforts, are capable of causing nutritive changes. Niemeyer believes that, when emphysema is due to forced inspiration, all the costal cartilages undergo degeneration; but that, when it is the product of forced expiration, only the uppermost costal cartilages are affected, and that, when the emphysema begins at a time of life in which ossification of the cartilages has already occurred, their hypertrophy and elongation, as well as fixed dilatation of the thorax, are impossible.

In case, then, we are able positively to exclude the etiological factors which have been mentioned as accompanying forced inspiration and expiration, and to recognize a fixation of the chest walls *before* the occurrence of the emphysema, we may take into consideration a primary fixed dilatation. But certainly this state of things is only very rarely met with, and therefore Freund's cartilage changes can be allowed only the weight of a complication and subsidiary cause.

General Features of the Disease.

As a rule, bronchial catarrh precedes emphysema by a year or more, and the catarrh may either be dry or be accompanied by a scanty mucous expectoration. It usually makes its appearance during the cold season, lasts through the winter, and disappears during the warmer months. In other cases the emphysema, under a rather acute form, is associated with some other disease of the respiratory organs, such as whooping-cough or pneumonia. Or, cough may be altogether wanting at first, as in kyphotic patients, in whom dyspnœa is the first marked symptom to show itself, followed by cough and subsequently by the further symptoms of emphysema. This is the more striking in cases of severe straining of the respiratory organs in connection with certain employments, such as playing on wind instruments, lifting weights, etc., and also in emphysema proceeding from an hereditary predisposition. In the latter two instances the emphysema often exists for a long time, and reaches a moderate

degree of development, before bronchitis supervenes secondarily, as the result of the altered relations of the circulation within the lungs.

Not infrequently the patients pass many years in succession without much annoyance, and their trouble first becomes marked with the increase of age and with the further spread of the emphysema in consequence of frequent and severe catarrhs or insufficient compensatory action of the heart, and may then increase to an unbearable degree. It thus happens that the general features of the disease show marked variations, depending upon the extent to which the affection is developed, and upon the complications and secondary changes in other organs.

It is usually the gradual occurrence of shortness of breath which first impels the emphysematous patient to call in the services of the physician. In the slighter grades of the affection the dyspnœa is moderate, however, and attracts the patient's attention only when heightened by the supervention of catarrh. But even now objective signs are to be recognized in connection with the respiratory organs. In other cases the patient complains of dyspnœa on brisk bodily movements, such as fast walking, running, and going up stairs. In still higher grades of the disease the dyspnœa occurs with any trifling movement of the person, such as a change of position, assuming the erect posture, etc., nay, it is even noticeable during repose or on speaking. The speech is often interrupted by the pressing need of breath, and, by reason of the concomitant bronchial catarrh, each inspiration is accompanied by a loud piping and wheezing sound. The voice is weak, and sometimes every word is brought out with a visible expiratory movement of the body. As a rule, even these patients feel better and more comfortable in the warm summer months, although their trouble clings to them year in and year out; and the agreeable, pure, and balmy air of the pine forests may even lead to a temporary condition of entire comfort. On the occurrence of cold weather, however, the shortness of breath returns in all its severity, and is markedly increased by the aggravation of the catarrh. The cough is dry and very tormenting, occurring especially in the evening and during the night, but also during the early morning hours, and

it may even assume a suffocative character. Expectoration is often, as has been mentioned, altogether absent, or there may be a sputum of a tenacious frothy character, brought up only by means of the severest straining and effort. It is only when the sputum appears in greater abundance and is more purulent that the dyspnœa is somewhat less intense, and the paroxysms of cough are less laborious and fatiguing. It is not uncommon for the dyspnœa to assume the form of asthma, consisting in attacks of terrible difficulty of breathing, which often for hours together throw the patient into a condition of the utmost anxiety for his life. Occasionally, and at the outset, these attacks appear at intervals of some months or weeks, but they are generally prone to increase in frequency, owing for the most part to an aggravation of the emphysema and of the accompanying catarrh, and finally, to the great torment of the patient, they recur every night. The breathing is hurried, and, on account of the squeaking and panting, noisy, and the expiration is very much prolonged. The patient is found sitting up in bed or in an arm-chair, with his arms supported and his body bent forwards in order to ameliorate the difficulty of breathing. He is highly cyanotic, the lips and hands being blue, the face livid, and the body covered with a cold, clammy sweat.

During the paroxysms of coughing the patient becomes cyanosed, the eyes protrude, and the jugular veins are distended. When the emphysema has lasted for a considerable time, and has reached a certain degree of development, the cyanosis becomes permanent. It shows itself at first only on the lips, the cheeks, and the nose, upon which enlarged and tortuous cutaneous veins are plainly noticeable. Subsequently the hands and feet also become cyanosed, and are habitually cold. Emaciation begins to take place, the skin becomes dry, the fat disappears, and the muscles, except those concerned in the act of inspiration, undergo atrophy. At the same time the strength is impaired, and the patient, very short-breathed under ordinary circumstances, is excessively fatigued by any sort of work. The neck is short and thick, and the jugular veins are markedly enlarged, their bulbs being often strikingly prominent. The sterno-cleido-mastoid, scaleni, and cucullares muscles are hyper-

trophied, shortened, and highly prominent. The supraclavicular fossæ are generally full, and on coughing they bulge out like air-bags. The thorax is enlarged in its dimensions, the upper part in particular being rounded, with a manifest increase of the sterno-vertebral diameter. The intercostal spaces are flat, but seldom prominent. The thorax is found in the permanent inspiratory position, and on inspiration it is drawn upwards *en masse* by the powerful action of the hypertrophied muscles of the neck, without the contractile action of the intercostal muscles being brought into play. Each quasi-spasmodic inspiratory elevation of the thorax is immediately followed by an expiratory sinking of the same, and in both acts the epigastrium is motionless, since, on account of the diaphragm having become useless by being pressed downwards, the inspiratory rising and expiratory falling of the epigastric region are, for the most part, wholly wanting. In case the diaphragm is pushed downwards to an unusual degree, so as to present a convexity looking towards the abdominal cavity, its contraction during inspiration may indeed produce a notable sinking in of the epigastrium. In most cases, however, distinct pulsations, synchronous with the radial pulse, are to be seen and felt in this region.

The apex beat of the heart is not generally to be discovered in its normal position. The area of cardiac dulness is small, it begins at a low level, and its lower limit, which can only be determined by ascertaining the position of the upper border of the right lobe of the liver, is, by reason of the depression of the diaphragm, situated one or two intercostal spaces below the normal level. The anterior and posterior limits of the lungs occupy a lower position, and the liver lies still lower, with its inferior border jutting forwards from beneath the costal arch. Although patients complain of palpitation of the heart, auscultation shows nothing abnormal about the orifices, unless there be complications and valvular lesions. The sounds are clear, but they are weak over the mitral, pulmonary, and aortic valves, by reason of the overlying portion of emphysematous lung ; on the other hand, they are stronger at the right border of the sternum, at the level of the fifth costal cartilage, where the heart is less overlapped by the lung. In case there is hyper-

trophy of the heart, the sounds are louder. Generally, too, the second pulmonic sound is notably intensified and accentuated in comparison with the second aortic sound. Percussion of the lungs elicits, as a rule, a loud and widespread resonance, corresponding to the increased area of the lungs. On auscultation we generally hear, besides the sounds proper to the accompanying catarrh, a weak vesicular murmur or very indeterminate breathing. The pulse is small and soft, the blood-pressure within the aorta is weak, and the urinary secretion is correspondingly scanty. The urine is of a red color, often showing a sediment of urates, and occasionally containing albumen.

It may take a year or more for the disease to attain a medium grade, and it may indeed remain stationary, to all appearance at least, for a long period, under the influence of appropriate dietetic and medicinal treatment. The patient is then in tolerable condition. Only the severer bodily movements give rise to dyspnœa, and during the inclement season of the year the aggravations of the bronchial catarrh which supervene cause him days of discomfort and nights of torment. He is therefore often obliged to keep his room for months together, and even, if there be febrile exacerbations of the catarrh, to remain in bed for a long time. Little by little, however, the gradually progressing emphysema increases in extent, new areas of the lung substance are constantly becoming unfit for the necessary interchange of gases, and the disturbances of the pulmonary circulation become more formidable and serve as the immediate occasion for an increase of the bronchial catarrh, which, by inducing severe paroxysms of cough, favors in its turn the extension and increase of the emphysema.

The dyspnœa increases and takes on the character of orthopnœa, with that feeling of constriction of the chest and that sense of impending death by suffocation which make night so particularly horrifying to the patient. The severest dyspnœa attacks him, and he is unable to maintain the horizontal position in bed, even with the shoulders decidedly raised. He manages to get through the night only by sitting on the bed or in a chair, with his limbs hanging down, so that the action of the auxiliary respiratory muscles may more freely be brought into play. The

dyspnœa is also intensified by a qualitative change, namely, that not only does the inspiration require the assistance of all the auxiliary muscles, but the expiration loses its merely passive character, and has likewise to be aided by a number of muscles. The breathing is therefore noisy; piping, wheezing rhonchi are audible at a distance, and are to be referred to a narrowing of the bronchi by the existing catarrh. The cyanosis reaches an extreme degree; the face, ears, neck, and limbs become dusky-blue, and even the tongue assumes this color. At the same time œdema shows itself—at first only around the malleoli. At this period the œdema is noticeable only in the evening, and, so long as the patient is still able to lie in bed, it disappears again during the night. It gradually becomes more pronounced, remains permanent, and occasionally gives rise to attacks of erysipelatous inflammation; it rises higher and higher until it reaches the inguinal region, when it attacks the scrotum, the labia, and the penis, and finally occurs also as a free effusion into the peritoneal cavity. Even the face becomes bloated, and assumes a yellowish-white or livid hue, while the lips, the cheeks, and the point of the nose retain their blue tint.

The pulse steadily becomes smaller, and at last irregular, the heart-sounds weaker and accompanied by murmurs, the urine scantier, more of a dusky color, and albuminous. The appetite fails, the bowels are confined, and hemorrhoidal excrescences, if they have not previously existed, form around the anus, which occasionally bleed, thus procuring the patient a measure of relief, although this is of but brief duration.

The patient complains of giddiness and of a roaring in the ears, and of headache, with a sense of pressure, and he becomes sleepy and apathetic. Meanwhile the dyspnœa constantly increases. Percussion discovers dulness of the dependent portions of the thorax on both sides, with diminished respiratory murmur and fremitus (hydrothorax). The sputa are exceedingly frothy, and in rare instances slightly streaked with blood; the râles become more moist and very abundant, both coarse and fine, and then the fatal termination—as œdema of the lungs has set in—is not far off. This result, besides being due alone to the emphysema and its sequelæ (fatty degeneration of the heart,

blood-stasis, and hydræmia), may be hastened, and the course of the disease modified, by complications and the intercurrence of other diseases, particularly the inflammatory, such as pneumonia. Death seldom occurs by cerebral apoplexy, or during an orthopnœal or asthmatic attack.

The foregoing applies only to substantive emphysema. On the other hand, the vicarious variety does not show symptoms so materially different as to constitute a special clinical history markedly at variance with that of the fundamental disease. Interstitial, interlobular, and subpleural emphysema show symptoms only when they have reached an advanced stage of development, and when a considerable quantity of air has collected in the pleural cavity. In such cases the percussion sound is very loud and sonorous, or tympanitic, the vesicular murmur is wholly wanting in the affected parts, and the dyspnœa increases to an extreme degree, and may speedily lead to death. If air escape through the mediastinum into the subcutaneous areolar tissue of the neck and breast, the well-known phenomena of subcutaneous emphysema appear—marked distention, puffiness, and pallor of the skin, which, besides, on palpation, gives the feeling of half-frozen snow.

Pathological Anatomy.

On opening the thorax of the highly cyanotic cadaver, by turning up the sternum and the costal cartilages, the imperfect retraction of the lung first attracts notice, the two lungs being in contact with each other at their anterior borders, and the pericardium, in the higher grades of emphysema, almost wholly covered in, particularly by the left lung, and crowded downwards and backwards away from the anterior thoracic wall. In cases of old pleuritic adhesions, marked hypertrophy of the heart, etc., this concealment of the organ may, of course, be wanting. The lung is enlarged in emphysema, often extending down as far as the seventh rib. The thorax is amplified by the depression and flattening of the diaphragm. The heart, which is situated at a correspondingly low point, lies in a more horizontal position, on account of the hypertrophy and dilatation of

its right side which are generally present, and is therefore displaced somewhat downwards and towards the median line. In case only one lung is affected, the heart may be pushed over to the opposite side.

In *vicarious* emphysema the lung is often enlarged (as in croupous pneumonia), or it may be of normal size or even diminished in bulk, since, by reason of antecedent diseases, interstitial pneumonia, shrivelling, or pressure from without considerable areas of lung tissue may be atrophied.

Even after the lungs are removed from the thorax they remain highly puffy and voluminous, with their borders rounded and not sharply defined. The reason why a healthy lung collapses only to a certain extent, without all the air being expelled from its alveoli, when the thorax is opened and the pressure of the outer air is thus brought to bear upon its surface, lies in the fact that the alveoli open into narrow, thick-walled bronchi, which speedily come in contact with each other on account of their weight, and thus hinder the escape of air. Now, in healthy lungs, it is true, the elastic alveolar walls overcome to a certain extent this impediment occasioned by overlying of the bronchi; but when, as in emphysema, the walls have lost their elasticity, and are even atrophied, the air escapes in only very trifling quantity, and the lungs retract only very slightly. If, along with the emphysema, there be also bronchitis, with swelling of the mucous membrane and obstruction of the bronchi by secretion, the air will not escape at all, thus rendering the puffiness still more marked.

Emphysema is generally *situated* at the apex of the lung and at the anterior borders, and occasionally in the tongue-shaped process of the left upper lobe, but it may be diffused over a large portion of the pulmonary surface, and is then more marked on the outer than on the inner surface. Less frequently we find emphysema extending deep into the lung. The *substantive* emphysema, due to heightened positive expiratory pressure with narrowing of the glottis, always has its seat in the upper lobes, and particularly in the apex. The *vicarious* emphysema, occurring in cases of wide-spread croupo-pneumonic infiltration, hypostasis, compression by pleuritic exudations, and tumors or

deformities of the chest, is generally situated at the anterior borders of the upper lobe, in the tongue-like process, or at the lateral borders of the base, often at a great distance from the original seat of disease, or even in the healthy lung. In cases of impermeability of a portion of lung, precisely those parts which, from their position in the front of the chest and on the diaphragm, are capable of the greatest possible amount of movement in the respiratory act, are most apt to act vicariously.

In partial obstruction of the bronchi and consequent atelectasis, in lobular-pneumonic infiltration, and in tubercular deposit, the distribution of the emphysema is very irregular, occurring in the form of circumscribed vesicles, from the size of a hemp-seed to that of a pea, grouped around each focus of the disease. Equally irregular also is its distribution when it is due to heightened negative inspiratory pressure, with primary nutritive lesion of the pulmonary parenchyma, since, in this instance, both its seat and its diffusion depend upon the original disease. Both lungs are generally affected, but sometimes one alone.

In *interlobular* emphysema, which has its chief seat at the anterior borders of the upper lobe, little air-sacs lie strung together under the pleura like a necklace, movable on pressure, and following fixed paths, corresponding to the boundary lines of the lobules. Interlobular may thus be distinguished from vesicular emphysema. Occasionally, however, these sacs are of more considerable size and more confluent, forming larger air-sacs, which raise the pleura from the subjacent pulmonary tissue. Bouillaud describes a case of this sort, thus far unique, in which a subpleural air-bag situated at the base of the left lung was, on account of its enormous size, at first taken to be the stomach filled with air. Rupture of the pleura, and the escape of air into the pleural cavity (pneumothorax) very seldom occurs, and still more rarely does it happen that the air beneath the pleura is forced towards the root of the lung, thence extending into the cellular tissue of the mediastinum, and, subsequently, into that beneath the skin of the neck or face (subcutaneous emphysema), or creeping downwards over the remainder of the chest.

Interlobular emphysema should not be confounded with the subpleural collections of air which are often found, in the form

of bladders of various sizes, in highly decomposed subjects and in blackish-green, putrid lungs. The latter constitute a cadaveric product, due to the development of gas in the interstices of the tissue.

The emphysematous lung is of a soft, down-like feel, like that of a cushion moderately filled with air, and yields easily to the pressure of the finger. The pits disappear, however, only very slowly, or, if the disease be very marked, they remain permanent. On incision, the air escapes gradually and almost noiselessly, or with a dry, hissing noise, quite different from the normal moist crepitation, and the walls collapse in proportion as they are not thickened.

The *color* of the lung in acute emphysema is pale red and anæmic, and the tissue dry. In the chronic form, abundant collections of black pigment traverse the white, very dry, and perfectly bloodless tissue, often in the form of delicate striæ, the remains of the blood-vessels which have previously undergone destruction.

Even to the naked eye the individual alveoli appear enlarged, in vesicular emphysema, to the size of a pin's head or that of a pea. But whenever, as is not uncommon, we find sacs as large as a bean or a hazelnut, or indeed of the size of a walnut or a pigeon's egg, which are occasionally separated from the surrounding lung tissue in the form of pedunculated roundish or oval bags, we may impute the condition to the formation of cavities, more or less like vesicles, by the confluence of a number of alveoli and lobules, due to atrophy and wasting of the alveolar and infundibular walls. It is customary to speak of a fine or a coarse vesicular emphysema, according to the size of the enlarged vesicles, but both forms are often met with together in one and the same lung.

On examining a section from a moderately distended and dried lung, we find the alveolar walls, in the lesser grades of emphysema, partly perforated, and partly changed by atrophy into delicate, low, shelving projections into the infundibular spaces. In the higher grades these projections entirely disappear, so that nothing but mere cyst-like forms remain, which may in turn, by still further atrophy, communicate with each

other. These cavities may either be connected with dilated bronchi, or they may be wholly independent of them. The walls of the most superficial vesicles are generally very thin, their tissue being merged in that of the pleura, or even formed from the latter alone. Occasionally the vesicular walls are thickened and stiff, and in the fresh condition do not collapse on incision. In our opinion this thickening is only apparent, being nothing else than the surrounding tissue compressed and blighted by the inflated vesicles. It seems doubtful to me if there is in these cases an actual hypertrophy or new formation of connective tissue and unstriped muscular fibres, as has been supposed by many, *e.g.* Wunderlich, Förster, Rindfleisch, and others, since I have not been able to demonstrate it to my own satisfaction.

Emphysema, whether it be due to inspiratory or expiratory pressure, begins—as I have observed in experiments on animals and in investigations of the human lung—with a rather moderate dilatation of the central infundibular space and of the alveoli which open into it. In addition, as Rosignol has stated, the bronchioles also become dilated. The alveolar walls are pressed against each other, undergo atrophy and thinning, even becoming perforated and wasted, and finally form, in common with the central infundibular space, large roundish-oval sacs, on the walls of which the former alveolar septa are now to be seen only as slight prominences. Several of these sacs, each corresponding to an original infundibulum and its alveoli, may lie in contiguity for a time. At last, however, their outer walls become thinned by the further effect of pressure, the sacs communicate with each other, and the walls become atrophied and almost wholly disappear, and thus from several of these sacs a large common cavity is formed, in which the original infundibular boundaries may still be recognized by the remains of atrophied septa and by the streaks of pigment deposited within them. At first the smaller sacs still remain connected with their bronchi, but, as they increase in size, the latter are made to disappear on account of the pressure, and, finally, the sacs may be wholly cut off from them.

The blood-vessels of the alveolar walls are, partly on account of the changes in the latter, and partly as a direct result of

increased air-pressure, changed into minute canals, scarcely pervious, and capable of admitting only the serum of the blood. On this account the blood-corpuscles accumulate in the vicinity of the narrowed portion, and even make their way through the walls of the vessels into the surrounding tissue, where they subsequently figure as pigment. The circulation soon ceases altogether, the neighboring vessels absorb the serum from the delicate canals, the vascular walls come into contact with each other, and, finally, after numerous fat granules have collected about the capillary channel, the vessels are obliterated. According to Klob, the wasting and closure of the vessels take place in consequence of excessive proliferation of connective-tissue cells in the adventitia. In the opinion of Isaakssohn, thrombosis by colorless blood-corpuscles is the cause, and fatty metamorphosis attacks the obstructing mass as well as the affected portion of the vessel. These changes in the capillaries react upon the veins and arteries, and may result in the destruction of a large extent of vessels. Under such circumstances, according to Rindfleisch, extensive anastomoses form between the pulmonary artery, the pulmonary vein, and the bronchial veins, by means of vascular arches which are peculiar in their great length, their equable diameter, and their want of branches; while, on the contrary, the numerous other arterial branches become highly tortuous and dilated pending the establishment of this communication. On account of this disturbance of the circulation, which is only partially remedied by the formation of the anastomosis, a not inconsiderable increase of lateral pressure takes place within the blood-vessels, plainly evidenced by the highly dilated vascular ramifications. This increase is extended to the trunk of the pulmonary artery, and leads to hypertrophy and dilatation of the right side of the heart, which we shall consider further on.

In the foregoing description of the microscopic changes in the pulmonary parenchyma in emphysema, the action of an absolute increase of air-pressure upon the alveolar walls, that is to say, of a purely mechanical force, has been assumed, as a consequence of which the existing textural lesions made their

appearance. But a primary nutritive etiological factor has already been mentioned, and no inconsiderable part ascribed to it in the development of pulmonary emphysema. We need not wonder, then, that many eyes have before now been directed to its discovery, and microscopic changes have occasionally been found, which have been pointed to with a certain degree of confidence as the tissue-change which predisposes to emphysema.

Rainey found fatty infiltration in the delicate membrane of the pulmonary vesicles. E. Wagner[1] first observed abundant aggregations of granules in emphysematous lungs. Villemin[2] believed that the cells found in the meshes of the capillaries became greatly enlarged in consequence of an inflammatory irritation, distending the walls of the alveoli, and increasing their capacity; that subsequently, on account of their further growth, the vessels were obliterated, and that after the degeneration and disappearance of the hyperplastic elements, the alveolar walls became attenuated. According to Wunderlich,[3] the walls of the pulmonary vesicles become thickened by catarrh, or by the long-continued vicarious action of a portion of lung, after the manner of the thickening of the bronchial mucous membrane in chronic bronchitis, and at the same time their capacity is increased; thus the adjacent vesicles, with their vessels, are compressed and obliterated, and the consequent impairment of nutrition leads to atrophy and wasting of the enlarged vesicles. As opposed to these observations, it is to be noted that, while fat globules are, to be sure, occasionally found in the capillaries and in the interstitial connective-tissue cells, they are, nevertheless, not necessarily to be looked upon as the cause, but rather as the result, of obliteration of vessels in consequence of the dilatation of the alveoli. As has already been mentioned, I am inclined to account for the thickening observed by Wunderlich as due to atrophy of the adjacent tissue; but in that case it is necessary to assume the previous existence of a tolerably well-marked degree of emphysema. The collections of proliferated nuclei seen by Wagner are but illusory, being due, according to O. Bayer, to

[1] *Uhle* and *Wagner*, Handb. der allgem. Pathologie, 2. Aufl., 1864, p. 386.

[2] Arch. Gén de Méd., Oct., 1866, pp. 385 et seq., and Nov., pp. 566 et seq.

[3] Handbuch der Pathologie u. Therapie, III., p. 421.

the fact that the nuclei, which are normally present, in equable distribution as regards size and number, in the inter-alveolar stroma, are now, by the emphysematous amplification of the alveoli, and by the distention of their walls, brought into one and the same microscopic plane, and thus seem to be more numerous.

Although investigation has not thus far led to any positive result as regards this question, still we cannot deny the existence of a primary nutritive lesion. The elastic tissue-elements of the lungs are the ones which are of importance in connection with the act of breathing, and in these, even when affected with actual lesions of function, no morphological changes are to be discovered. A *tissue-relaxation*, then, may be present in the lung, without our being able to recognize any corresponding microscopic abnormality. According to Bayer, such a disturbance in the function of this tissue may depend upon modifications in the circulation, and emphysema may thus be produced in lungs which are otherwise diseased, or in the course of affections of other organs which involve certain derangements in the pulmonary blood circuit.

Moreover, diseases in the lung and in other organs—such as bronchitis, tuberculosis, cirrhosis, atelectasis, catarrhal and croupous pneumonias, extensive pleuritic adhesions, diseases of the larynx, etc.—occur in conjunction with the emphysema, and either take place as accidental complications, or stand in a causative connection with it, or indeed are to be considered as its consequences.

Among the *consequences*, as regards diseases of the bronchi (apart from dilatations, constrictions, and obliterations), belongs *bronchial catarrh*, which, to be sure, is generally the primary lesion, but the secondary occurrence of which is nevertheless, according to what we observe in the emphysema of children and in certain cases in adults, not to be overlooked. A substantive emphysema due to other causes than primary bronchitis will, then, by reason of the circulatory disturbances above mentioned, necessarily lead to hyperæmia of the bronchial mucous membrane in healthy portions of the lungs, and thus occasion catarrhal diseases.

The *heart* becomes hypertrophied, as the immediate conse-

quence of the increased pressure in the pulmonary artery due to the destruction of numerous pulmonary capillaries and the imperfect establishment of anastomosis. The right ventricle and its conus arteriosus are not specially thickened as regards their walls and the columnæ carneæ. The auricle is dilated, and filled with an abundance of dark blood. The heart is of a roundish form, its apex springing from the right ventricle; it lies more horizontally than natural, and it is turned on its long axis from right to left. Its muscular tissue is at first tough and firm, and of a brownish-red color; but in the subsequent course of the affection it may become flabby and of a pale grayish-red, or it may even assume a yellowish punctate or striate coloration, as the result of simple granular or fatty degeneration. The preceding deficient nutrition of the heart, due to disturbances in the pulmonary circulation and to diminished decarbonization, in connection with its over-activity, unquestionably favors degeneration of its substance. This leads to imperfect compensation; over-distention and dilatation of the coronary veins occur, with hydropericardium. On account of the impeded return of the blood from the venæ cavæ, static hyperæmia of the *brain*, *stasis in the systemic venous circulation*, stasis in the *hepatic veins*, and, in consequence of atrophy of the central liver-cells, *cyanotic atrophy of the liver*, or atrophic cirrhosis, may occur. Hence, also, on account of stasis in the portal system, hyperæmic and chronic *catarrhal conditions* of the mucous membrane of the *stomach* and *bowels* take place, together with enlargement and hardening, and even new formation of connective tissue in the *spleen*, which is engorged and of a marked brownish-red color. Subsequently we find engorgement of the *kidneys*, with enlargement at first, followed by a diminution in the size of the organ, and wasting from proliferation of connective tissue, together with *albuminuria*. Associated with this state of things there may be *hydrothorax*, *ascites*, *anasarca*, and *œdema of the lungs*.

If the venous stasis be extreme, the increased blood-pressure does not seem to be wholly spent upon the capillaries, but to extend also to the arteries, and thus to occasion such an increase of pressure in the aorta and the left side of the heart, together

with heightened activity of the latter, that the left ventricle likewise becomes hypertrophied. It may also become weakened and diminished in functional energy. Lebert found hypertrophy of the thyroid gland six times among twenty-one cases of emphysema in Zürich, and believes it to be a contributory etiological factor. The upper part of the thorax is generally enlarged in a barrel-shaped manner, and the muscles of that region (the scaleni and the sterno-cleido-mastoids) are markedly hypertrophied. The ribs and the costal cartilages are rigid and thickened, and the latter even ossified. The dependence of these thoracic changes upon nutritive lesions of the cartilages, through the agency of distortion or hyperæmic conditions of the chest walls, has already been mentioned.

Relations with other Diseases.

It has always been considered that there were certain special relationships between individual diseases, by virtue of which the existence of one precluded the subsequent development of another. Rokitansky maintained that there was a relationship of this sort between emphysema and tuberculosis, because the cardiac enlargement and venous stasis (the "venous crasis" of Rokitansky) occasioned by emphysema seemed capable of affording protection against tuberculosis. By others the immunity from tuberculosis was attributed to the bloodless condition of the apex of the lung. Ramadge is of the opinion that tuberculosis may end in recovery, in consequence of the development of an emphysema, and Frey even proposes the artificial production of emphysema for the purpose of curing tuberculosis. On the other hand, Dittrich speaks of the emphysematous enlargement of the pulmonary alveoli as favoring the formation of tubercle, and that such a formation is checked by the diminution in size of the pulmonary cells. Careful observations have shown that such an immunity does not exist to the extent which has been claimed by authors. Persons affected with emphysema do not, indeed, often contract tuberculosis, but they may be attacked with acute miliary tuberculosis as well as with chronic tubercu-

losis,[1] and, moreover, vicarious emphysema may be associated with any form of tubercular disease.

Lesions of the cardiac valves are seldom met with in emphysema, and we must therefore conclude that emphysematics are only slightly liable to valvular endocarditis and endarteritis, although, according to Chambers and Biermer, the latter occurs more frequently than the former. On the other hand, Lebert,[2] in autopsies of emphysematics, found valvular lesions to have developed after emphysema in ten per cent. of the cases—mitral insufficiency and retraction in eight cases, aortic lesions in two cases, and affections of both the left orifices in one case. Slight emphysema may occur secondarily in cases of valvular lesion. *Inflammations* likewise, particularly the croupous, seldom occur in the course of emphysema. Some ascribe this to the venous blood-crasis, and others to the obliteration of vessels.

Analysis of Individual Symptoms.

Substantive Emphysema.

The enlargement of the chest in all its dimensions is produced by certain muscular actions in quiet *inspiration.* Thus, its length is increased by the contraction and descent of the diaphragm, its depth by the elevation of the ribs, and its breadth by the rotary movement which they perform and which results from the contraction of the external and internal intercostal muscles. *Expiration* occurs upon the cessation of these muscular actions, the previously dilated thorax resuming its former shape by virtue of its elasticity and the ascent of the relaxed diaphragm. The lung follows the chest in its expansion. It completely fills the increased space occasioned by the dilatation of the chest, and on the cessation of the latter it contracts to a certain size by reason of its elasticity and tension, thus expelling a certain quantity of air. That the lung exerts no positive action upon the sternum and ribs in the act of expiration, as aiding their resumption of the inspiratory position, is plainly apparent,

[1] *Büchner's* case, Würzb. Zeitschr., II., 1.

[2] Klinik der Brustkrankheiten, I., p. 377.

and this seems to me to be true also in the case of children, although Niemeyer admits the possibility of such an action in them. But upon the diaphragm the elastic lung exercises a traction which certainly is not to be estimated as inferior to the upward pressure of the abdominal organs. According to Niemeyer, this statement is corroborated by the fact, that in the dead body the diaphragm is always arched upwards, even when the walls of the abdomen are perfectly relaxed or its contents removed, provided only the thorax be still entire. It sinks downwards, however, as soon as the latter is opened, since the force exerted by the lungs then comes to an end. But whenever the elasticity of the lung is diminished or altogether lost, and the lung remains in a permanent inspiratory position, the air being forced out from it in very inconsiderable quantity, then the negative pressure, *i.e.*, the traction on the diaphragm, fails. Therefore the diaphragm cannot rise and resume its expiratory position.

Thus, *the first and most important disturbance* in emphysema is *difficult expiration;* whilst inspiration, in so far as regards the act itself and the forces which accomplish it, remains unaffected. The latter may even be easier than usual, owing to the diminished elasticity and tonicity of the lungs, which, together with the thoracic resistance, have to be overcome by the muscles in the act of inspiration. But in natural, quiet breathing, as is well known, the air is not entirely expelled from the lungs in expiration, but a certain residuum is always left, which is increased in quantity in direct proportion to the diminished action of the pulmonary parenchyma, that is to say, to the impairment of its elasticity. The greater is this reserve in quantity, the smaller will be the amount of new air to enter on inspiration, so that, in spite of the unimpeded efficiency of the inspiratory muscles, the effect of the act is in nowise heightened, but rather impaired, and thus a disturbance of inspiration may follow secondarily upon that of expiration.

On this account the patient has a sense of deficiency of air, a sort of longing for air [Lufthunger], and a feeling of oppression, and, in order to assuage these sensations, the act of inspiration is intensified by means of the auxiliary inspiratory muscles.

In order to elongate the thoracic space, in spite of the inadequate descent of the diaphragm, the muscles at the upper part of the chest, the sterno-cleido-mastoidei and the scaleni, are called into action, and by means of their contraction, the head and the cervical vertebræ being fixed, the thorax is drawn upwards. In the more pronounced grades of dyspnœa, the erector muscles of the spine act as inspiratory muscles, since they also elongate the thorax; and so, likewise, do the levator muscles of the ribs, the shoulders, etc., which, arising partly from the shoulder-blade and partly from the arm, are inserted upon the thorax. The action of these muscles is then the direct opposite of their normal function—fixing points which they usually move, *i.e.*, the head, the shoulder-blade, and the arm; and moving the thorax, which is ordinarily fixed. They are therefore of service, under these circumstances, only when aided by fixation of the normally mobile points by extrinsic means, such as leaning upon the arms, etc. From being diaphragmatic the inspiration thus becomes entirely costal. But the diaphragm, if it be very much depressed and arched downwards, may even ascend on contracting, and thus contribute to expiration rather than to inspiration. If a number of bronchi in the lower lobes be so blocked up by secretion as to impede the entrance of air, these labored inspiratory efforts may cause retraction of the lower portions of the thorax. In the severer cases of dyspnœa, with a fixed and rigid thorax, and ossified costal cartilages, all elasticity is wanting, and there is no sinking of the chest-wall on expiration. Under such circumstances, even the expulsion of but a small portion of air from the lungs has to be accomplished by certain positive muscular forces which do not come into play in the normal quiet act of expiration. The abdominal muscles are then thrown into powerful contraction, so as to act upon the contents of the abdomen, and, through these, upon the diaphragm. In other instances, the patient, who has lain at full length during inspiration, bends the upper part of his body forward during expiration, thus forcing the diaphragm up by compressing the abdominal contents, or he grasps the lower portion of the chest with his hands, or rests on his hands and knees in bed, and in this way facili-

tates inspiration, while assisting expiration by pressure on the abdomen.

The embarrassed breathing of emphysematics does not result solely from the loss of elasticity in the lung-substance, and from the permanent inspiratory state of the thorax, but it is also in great part owing to the wasting of the alveolar walls, and to the accompanying obliteration of a considerable number of pulmonary capillaries. It is self-evident that the extent of pulmonary surface capable of serving to absorb oxygen and to eliminate carbonic acid is thereby diminished. It must be true, too, that the dyspnœa of emphysematics is increased in no trifling measure by concomitant bronchitis, with swelling of the mucous membrane, and plugging of the smaller bronchi with a more or less tenacious secretion.

In addition to the persistent shortness of breath, attacks of *asthma* often occur, which are attended with a striking prolongation of the expiration, and with hissing noises, which not infrequently recur regularly at certain hours of the day, and which are to be referred to spasm of the bronchi. They are of reflex origin, and generally due, as Biermer believes, to accumulation and tension of the residual air. Such attacks may also be occasioned by acute intercurrent bronchial catarrh, by flatulence and indigestion, by derangement of the circulation of blood in the abdominal organs, by the inhalation of dust, and by emotional disturbances.

Intimately associated with the disorders of the respiration are those of *the circulation.* These are in part due to the *destruction of numerous pulmonary capillaries*, and in part to the *diminished suction-power of the lungs*, resulting from loss of the elasticity of their parenchyma.

It has been established by Donders that the pressure of air upon the vessels within the lungs is equal to the external atmospheric pressure of a column of mercury 760 millimetres in height, minus the elastic force of the lungs, which is equivalent in ordinary expiration to that of a column of mercury of 7.5 millimetres. Hence, even between the acts of respiration, a suction force is exerted upon the blood-vessels within the chest, especially upon the bulky and thin-walled veins. At each inspira-

tion the elastic tensile force of the lung is increased—ordinarily to eight or nine millimetres of mercury, and, on deep inspiration, to thirty or forty millimetres—and thus, of course, the suction is intensified. With each expiration the vessels undergo a considerable pressure, which, in placid breathing, serves to assist in emptying the arteries; but which, on the other hand, in forced expiration, impedes the entrance of blood into the thoracic veins. If, now, the inspiratory and expiratory acts, and the tensile force of the pulmonary parenchyma, are reduced to a minimum in emphysema, the difference between the external and internal air-pressure will be correspondingly reduced, and the suction-power of the lung will fail to affect the circulation. On account of the persistently heightened and unvarying air-pressure to which the thoracic vessels are subjected, the entrance of blood into the great cavities and into the heart is impeded, the great venous trunks become overfilled, and the lungs are insufficiently emptied of blood.

The latter condition, together with the destruction of a number of the pulmonary capillaries, leads to collateral hyperæmia of those portions of the lung which are free from emphysema, to bronchitis and pulmonary œdema, as well as to over-distention of the pulmonary artery and right side of the heart. The already pre-existing dyspnœa is thus increased in no inconsiderable degree. These disturbances may, however, be very decidedly diminished by the compensatory *eccentric hypertrophy of the right ventricle*, which arises in consequence of the heightened blood-pressure, so that such patients are often only slightly incommoded by the emphysema while they remain quiet, and only complain of shortness of breath on brisk bodily movements, such as fast walking, going up stairs, and the like.

It is only when the heart becomes weakened, in consequence of granular, fatty, or amyloid degeneration of its primitive muscular fasciculi, that it is no longer able to overcome the increased resistance to its action, and then the hypertrophy gives place to passive dilatation,[1] which is accompanied by a train of addi-

[1] No considerable degree of hypertrophy of the right ventricle can long be maintained in cases of emphysema, since heightened functional activity of an organ calls for a corresponding increase of its nutritive supply. Now, the regular and adequate

tional symptoms referable to stasis in the superior and inferior venæ cavæ and their tributaries. The jugular veins swell, and may undulate with each ventricular systole; the frontal and temporal veins are highly distended, often forming thick cords under the skin. The lips, ears, alæ nasi, and cheeks become cyanotic, and their vascular ramifications appear varicose. In the highest grades of *cyanosis* the color is a dark bluish-red, resembling that formed upon the cadaver, whilst the skin of the remainder of the face is pale and livid from a paucity of blood in the arteries. The same cyanotic coloration is shown by the hands and feet, particularly at the roots of the nails; both of these parts are constantly cold, and, on account of the sluggishness of the circulation and the deficient access of arterial blood, the patient finds it difficult to warm them.

Hyperæmia of the brain, which, particularly during the paroxysms of cough, becomes excessive, and may, on occasion, lead to apoplexy, is marked by headache, a sense of pressure in the head, dizziness, roaring in the ears, and muscæ volitantes. The patient subsequently becomes drowsy, and incapable of mental exertion, whilst the facial expression is sullen or apathetic, or plainly reveals the patient's anxiety and oppression. The impeded flow of blood through the inferior vena cava leads directly to *œdema of the feet*, especially about the ankles, which is most marked in the evening, after the patient has gone through the exertions of the day, and disappears again during the night. Patients complain also, especially on coughing, of pain in the right hypochondrium, due to swelling of the liver from static hyperæmia. In consequence of the retardation in the portal circulation, this is accompanied by a feeling of fulness and pressure in the epigastrium, a deficient appetite, pyrosis, impaired digestion, constipation, and tympanites, due to *gastro-intestinal catarrh*, as well as by varicose dilatations of

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provision of the latter through the coronary arteries is precluded by the obliteration of numerous pulmonary capillaries, which leads to stasis in the heart, with serous infiltration into its tissue, in consequence of deficient emptying of the coronary veins into the right auricle. Both of these occurrences are capable of speedily abolishing the compensatory action of the heart by simple weakening or degeneration of its muscular substance.

the rectal veins, blind or even bleeding piles, which latter may, to a certain extent, relieve the sense of distention in the abdomen.

On account of the deficiency of blood in the aortic system, and the diminished pressure, the *pulse* is small and weak, and in case of cardiac degeneration it is sometimes irregular and intermittent. For the same reason the *urine* is scanty and dark-colored, rich in solid constituents, and of higher specific gravity than normal. Sediments form, partly from the deficiency of water, and constitute the well-known brick-red deposit of urates. The latter are, moreover, absolutely increased in quantity, owing to the disturbance of respiration, and to deficient oxidation and conversion of uric acid into urea. The quantity and composition of the urine usually vary greatly during the course of the disease. In moderate emphysema, where the collateral pulmonary channels suffice for the requisite filling of the left side of the heart, the urine is normal, but in severer cases its quantity is diminished. The latter may, however, gradually become normal again on account of compensatory hypertrophy of the right ventricle. It is only on the occurrence of weakening, degeneration, and deficient activity of the heart that the urine is markedly diminished in quantity, and even then the secretion may, upon stimulation of the heart's action, occasionally be increased, although but for a short time. In cases of excessive stasis in the kidneys we not infrequently find albumen, blood, and even fibrinous casts, together with cast-off epithelium, which has partly undergone fatty degeneration. Furthermore, it is merely necessary to allude to the fact that the quantity of solid constituents depends upon the nutritive condition of the individual.

The digestive disturbance attendant upon the gastric and intestinal catarrh, and still more the inadequate emptying of the thoracic duct into the left subclavian vein, over-distended with blood, lead to impoverishment of the blood in so far as concerns such of its elements as are derived from the lymph (colorless blood-corpuscles, fibrine, and albumen). Add to this, too, the insufficient decarbonization of the blood in the lungs, and we can readily understand the *general mal-nutrition* which soon attacks

the emphysematic. The patient loses flesh, the skin becomes dry, and the muscles of the limbs become atrophied in contrast with the hypertrophy of the respiratory muscles. According to Walshe, it would seem also that the specific gravity of the body is diminished. Thus the patient becomes debilitated, his locomotion is embarrassed, his movements are laborious, and his attitude stooping. The above-mentioned impoverishment of the blood favors the *dropsical phenomena*, which are primarily due to circulatory disturbances. The eyelids swell, and the face becomes bloated. As the dropsy commonly travels from below upwards, it is most marked in the lower extremities, which often attain an astonishing bulk. It then attacks the scrotum, the labia majora, the prepuce, the abdominal walls, and, on dorsal decubitus, even the soft tissues of the back. Effusions into the serous cavities may also be present, such as ascites, hydrothorax, and hydropericardium, which, as a matter of course, increase the dyspnœa to an extreme degree, so that the patient cannot lie in bed at night, or even maintain a semi-recumbent posture, but is obliged to lean against a chair.

Cough and *expectoration*, which ordinarily accompany emphysema, belong not to the emphysema, but to bronchitis. The former is generally dry, deficient in force, and often very tormenting, and in its severer attacks it occurs especially in the evening and at night. If there be any sputum, it is usually scanty, tenacious, and slimy, seldom thick and yellowish, and, whenever such complications as bronchorrhœa, bronchiectasis, etc., occur, it is opaque, or even entirely purulent. Streaks of blood in the sputum, or a considerable quantity of clear blood are rare, occurring only from rupture of vessels after severe paroxysms of cough. Whenever blood is coughed up for days together, and without the cough being attended with excessive straining, we may suspect the existence of hemorrhagic infarctions.

Physical exploration reveals nothing in the slighter cases of emphysema, but in the higher grades of the affection, however, it brings out very important diagnostic points.

Inspection and *Palpation.*—1. The thorax may be normal, and devoid of any specially characteristic deformity, or it may

be long and flat, constituting a true "paralytic" thorax. 2. More commonly it is enlarged in all its dimensions with tolerable uniformity. 3. Most commonly the enlargement is but partial, and it particularly involves the upper and middle portions in their transverse and antero-posterior diameters, whilst the lower part of the thorax is either contracted, normal, or, rarely, somewhat enlarged (the "barrel-shaped thorax"). The occasional dilatation of the lower portion of the thorax is explained by the liver having been pressed downwards, whereas the more frequent narrowing is to be referred to the action of those abdominal muscles which come into play in forced expiration, and which by their contraction draw the lower ribs inwards as well as downwards.

These three forms of thorax may be explained without special difficulty. If emphysema attack the lung tissue at a time when the costal cartilages are already fully ossified and incapable of assuming any marked change of form, the thoracic wall will not be affected in its configuration. The characteristic form of the thorax in emphysema is, then, dependent upon the yielding condition of the costal cartilages, and upon a change in their shape.

The strain upon the costal cartilages in deep inspiration and expiration, together with the circulatory disturbances occasioned by the emphysema and those of its causes which reside within and around the chest, leads to changes in the nutrition of the bony and cartilaginous portions of the ribs, with increase in their thickness and length. By their elongation the sternum is carried further forwards, and thus the antero-posterior diameter of the thorax is augmented. By the outward and upward bowing of the ribs, which takes place at the same time, resembling the motion which occurs at each inspiration, the thorax becomes increased in breadth, and thus a permanent change of form is brought about. If the emphysema be due to forced inspiration, this change affects the whole thorax, because in such cases all the ribs and cartilages alike undergo the strain; whilst, if the emphysema proceed from forced expiration, the change is confined to the upper and middle portions of the chest, since then, according to Ziemssen's observations, we can only take the upper ribs into account. In the latter case, the so-called "bar-

rel shape" of the thorax is produced, its occurrence being the most common simply because emphysema most frequently originates from forced expiration. In emphysematous dilatations of the thorax, the sterno-vertebral diameter is always chiefly affected, since the sternum most readily yields to the pressure of the elongated ribs. Even the so-called inspiratory dilatation of the whole thorax is never uniform, but more pronounced at some parts than at others, which fact is doubtless due to an unequal change in the cartilages.

Thus are explained the thoracic changes adduced by Woillez: 1. A total arching forwards of individual ribs over the whole anterior and lateral regions of the chest, more commonly on the left than on the right side. 2. A prominence extending from the sternum to the nipple (sterno-mammillary). 3. A convexity reaching from the clavicle to the nipple (cleido-mammillary). 4. Bulging of the supra-clavicular fossa (supraclavicular).

Therefore, in our opinion, the abnormal form of the thorax is dependent upon the cartilage tissue being altered in its normal nutritive conditions by strain and circulatory disturbance, as well as upon the changes in form and position produced by forced inspiration and expiration in the cartilages thus relaxed. At the same time it is not to be disputed that forced expiration, with the glottis closed or narrowed, may alone, by means of heightened intra-thoracic pressure, produce alterations of form in a soft, yielding thorax. For such a change of shape to become permanent, however, a secondary nutritive lesion of the cartilage must certainly supervene.

The intercostal spaces are wide and flat in emphysema, the muscles flabby, but capable of action and not bulged out. The clavicles are markedly curved, and the same is true of the spinal column in its lower dorsal and upper lumbar portion. The shoulder-blades stand out from the chest like wings. The neck is thick in front, on account of the great development of the muscles which serve to raise the thorax—the scaleni, the sterno-cleido-mastoidei, and the cucullares; it is short, and directed markedly forwards, so that the jugular and lateral fossæ appear deepened.

The thorax is only slightly raised at each inspiration, even if it be forced; the scaleni and the sterno-cleido-mastoidei become

shortened, thus still further deepening the jugular and supra-clavicular fossæ. In high degrees of emphysema, with a stiff thorax, the effect produced by the intercostal muscles amounts to nothing at all. The epigastrium and the sides of the abdomen are generally puffed outwards, and, by reason of the diminished action of the diaphragm, show less motion than normal, whilst the middle and lower portions of the abdomen are pressed forwards. With each expiration, too, the thorax sinks only to a limited extent, the supra-clavicular fossæ become flatter, and occasionally they are wholly obliterated. The veins of the neck are noticeably prominent, and show evident wave-like motions. The abdominal muscles become very tense, causing retraction of the abdomen. In the middle of the abdomen we find a tolerably deep transverse furrow, which corresponds to a certain extent with a horizontal line extending from the junction of the twelfth rib with its cartilage to the same point on the other side. This results from an inordinate tension of the upper portion of the transversalis muscle, which takes its origin from the inner surface of the six lower costal cartilages, which it tends to drag downwards, so as to contract the thorax. In contrast with this marked tension of the upper portion is the feeble contraction of the lower portion of the same muscle, which arises from the transverse processes of the upper lumbar vertebræ, from the lumbo-costal ligament, and from the crest of the ilium. Consequently this furrow is observed only when the thorax is perfectly immovable. Occasionally, on forced and impetuous expiration, as in coughing, we see the supra-clavicular fossæ swell out like bags. So long as this phenomenon involves only the inner portion of the fossa, it may, as Niemeyer believes, proceed from excessive distention of the bulb of the jugular vein; but when the outer part of the fossa, towards the acromion, bulges out, it depends upon the forcible upward impulsion of the apex of the lung, which may readily be recognized in a thin person with an atrophied condition of the platysma myoides.

On *palpation*, the hands laid upon the chest are, on account of its diminished motion, only slightly raised. The *pectoral fremitus* is normal, or, on account of impaired vibratory capacity of the thorax and feebleness of the voice, diminished. In the

epigastrium we see and feel a tolerably strong pulsation, which is often communicated to the lower portion of the sternum and to the corresponding costal cartilages. This does not arise from the apex-beat, but from a systolic impulse on the part of the right side of the heart, which, being hypertrophied, and occupying a transverse position, imparts its motion to the diaphragm and to the left lobe of the liver. The true apex beat is seldom felt, owing to the heart being so far separated from the thoracic wall by intervening lung tissue. When it is felt it is weak and does not occupy its normal situation, but is displaced downwards to the extent of one or two intercostal spaces by the depression of the diaphragm, and several fingers'-breadths outwards, owing to the horizontal position of the heart.

Percussion generally elicits a very loud sound, which, however, may be rendered somewhat less sonorous by marked fixedness of the thoracic wall, and by an abundant muscular covering. A tympanitic percussion sound is not produced as the result of mere emphysema, but occurs only in certain complications associated with a loss of tonicity in the lung tissue. So long, then, as the lung tissue possesses a certain degree of tension, as is always the case in emphysema, the alveolar walls are collectively thrown into vibration on percussion, which interferes with that regular vibration of the air within the vesicles which is requisite for the production of the tympanitic sound.

Biermer[1] found, particularly at the lower and lateral regions of the thorax, a special modification of the sonorous percussion sound, which, from its resembling the sound produced by striking on a band-box, he calls, "bandbox sound" (Schachtelton). This sound is deep and sonorous, with a somewhat tympanitic reverberation. Biermer thinks that it depends upon a very high tension of the alveolar tissue, and that it chiefly occurs in situations where there is a pretty extensive contact of distended portions of lung with the thoracic wall. I have occasionally found a similar percussion sound also on the anterior aspect of the chest in cases of large emphysematous sacs lying in immediate contact with comparatively very thin thoracic walls. I conceive it to be produced in this wise: the great mass of air occupying a superficial position comports itself in very much the same way as would a similar mass, situated outside of the lung and confined within a sac, when thrown into regular vibrations by percussion; that is, the vesicular walls are, from

[1] Handbuch, p. 809. Volkmann's Vorträge, p. 48.

their tenuity, unable to interfere with these regular vibrations. The compression of the adjacent tissue, which has already been mentioned as due to pressure on the part of large sacs, may also have an influence in this respect. If that be the case, a tympanitic reverberation will thus be added to the highly sonorous tone.

According to the extent of the lung the loud percussion sound exceeds the normal limits, and reaches, on a line through the nipple, to the seventh or eighth rib, or even to the lower boundary of the ribs, so that the hepatic dulness begins there only; behind, it extends to the tenth, eleventh, or twelfth rib. Notwithstanding the existence of hypertrophy of the heart, the area of cardiac dulness is generally very much curtailed by the intervening lung, and in the higher grades of emphysema it may be wholly wanting. It commonly begins, owing to the low situation of the diaphragm, at the fifth or sixth rib, is bounded on the right by the left border of the sternum, and extends toward the left to about midway between the sternum and the nipple. Its lower limit is difficult to define, since it runs into that of the left lobe of the liver, and since the apex is generally neither to be seen nor to be felt.

On *auscultation* we find, in contrast with the loud and clear percussion note, a weakening of the vesicular murmur, proportioned to the degree of the emphysema and to the more or less imperfect play of air in the alveoli and bronchi, and occasionally it is almost entirely wanting over the whole extent of the lungs. Even the normal bronchial breathing over the course of the great bronchi, between the shoulder-blades, may be altogether absent. At the front of the chest we sometimes hear a prolonged, very exaggerated, and, in the words of Niemeyer, "whizzing" (zischendes) inspiratory sound. I have met with it in cases of emphysema due to positive inspiratory pressure, in which the apex of the lung was highly dilated, while the anterior and lateral portions were only slightly emphysematous. Under such circumstances there is no movement of air in the upper portion of the lung, whilst the anterior and lateral portions, which still remain healthy, seem to receive in a vicarious manner the air which pours into the lung, especially if catarrh have produced narrowing and obstruction of the bronchi behind and below. On forced inspiration, in such cases, the air enters the alveoli with a certain

impetus, and produces the sound. In the further progress of the emphysema this phenomenon may disappear little by little, which is indicative of a gradually increasing inelasticity of even this portion of the lung. Lebert describes, in addition, a peculiar, parchment-like crackling ("alveolar crackling") occasionally heard on inspiration in the upper and anterior part of the chest, the cause of which would seem to lie in the forcible friction and distending action of the air in the dry and dilated alveoli on strained inspiration.

There is no expiratory sound, unless the emphysema be accompanied by bronchitis. The frequency of the latter, as a cause or as a complication of emphysema, is apparent therefore from the fact that in the majority of cases a distinct and prolonged expiratory sound is audible—due to narrowing of the bronchial tubes. Together with this we find still other catarrhal sounds, which are of a drier character from the frequency of dry catarrh as a complication. Oppolzer mentions also a dry, coarsely vesicular râle, heard towards the close of inspiration, in cases where the emphysematous vesicles are enlarged to the size of a bean; this râle he compares to the sound produced by "smacking with the tongue."[1]

The cardiac sounds are weak over the mitral valve, and over the aorta, in consequence of the overlying lung; but they are well marked at the right lower border of the sternum, and at the epigastrium. The second pulmonary sound is rendered more distinct and accentuated by the increased pressure existing in the pulmonary artery, but it becomes weaker again in cases of degeneration of the heart substance and of insufficient distention of the pulmonary artery.

The murmurs which are sometimes heard over the valves in emphysematics, particularly over the auriculo-ventricular openings, have received various explanations. Gerhardt[2] attributes the production of the sound to the mitral valve, and believes it to be of an anæmic nature, due to the left ventricle being insufficiently filled with blood, by reason of the emphysema. On the other hand, Biermer maintains that the phenomenon occurs even

[1] The *râle crepitant sec à grosses bulles* of *Laënnec?*—TRANSLATOR.

[2] Lehrb. d. Ausc. u. Percuss., 1. Aufl., p. 247.

in cases of perfect filling and hypertrophy of the left ventricle, and considers that it depends upon a relative insufficiency of the tricuspid, since in some cases pulsation of the cervical veins is also met with. Both are unquestionably correct; yet, on the one hand, cases of well-marked emphysema occur, with very insufficient filling of the left ventricle, in which the sound is absent; and, on the other hand, there are cases in which, with a visible apex beat, the sound is more marked over the mitral than over the tricuspid, and must therefore be referred to the former, while yet the emphysema is not extreme and the deficiency of blood in the left ventricle is not very pronounced. In such cases we find on post-mortem examination, and with the aid of the microscope, either fatty degeneration of the papillary muscles and of the cardiac walls, or merely a very abundant, finely granular cloudiness of the muscular fibres of the left side of the heart—the expression of a nutritive lesion due to the emphysema or to simultaneous general causes.

Vicarious emphysema, if of considerable extent, resembles the substantive affection in its symptoms, which, however, generally make their appearance less frankly, or are concealed or modified by the phenomena of the fundamental disease. But, if the emphysema be of but moderate extent, symptoms will be wholly wanting. At most, it will betray itself at the anterior borders of the lungs, by the existence, over a circumscribed portion of the normal area of cardiac dulness, of a loud or tympanitic resonance, which immediately runs into the somewhat irregularly shaped area of cardiac dulness.

Interlobular and *subpleural* emphysema runs its course without any characteristic symptoms; for Laënnec's statement, that it gives rise to a particularly full percussion note, and to a dry friction sound, produced by the rubbing of air sacs, which stand high above the general surface of the lung, against the costal pleura in the movements of respiration, has proved to be incorrect. It is only when the emphysema travels from the root of the lung into the mediastinum, and from thence into the subcutaneous tissue of the neck and rest of the body, that the symptoms become significant and admit of the recognition of the disease at a glance. The air collected in the mediastinum

presses upon the heart and great vessels, and occasions suffocative attacks of the greatest severity. For the rest, the skin is intensely white and glistening, and puffed up like an air-cushion; it crackles under slight pressure with the finger, and yields a tympanitic sound on percussion. The patient complains of pain in the parts distended with air, which, however, is not particularly increased on motion.

Duration, Results, and Prognosis.

Duration.—Interlobular emphysema is always acute in its rise and progress. This is seldom the case in the vesicular form, as after whooping-cough and bronchitis in children. Ordinarily it is of gradual development and runs a slow course, frequently lasting for a term of five, ten, twenty, thirty, or forty years. Indeed there are cases in which emphysema is developed in early life, and yet the patients reach a ripe old age.

Results.—The milder grades of vesicular emphysema, or rather of distention of the pulmonary parenchyma, such as are sometimes found in autopsies after acute bronchitis, pneumonia, or whooping-cough, may disappear in the course of a lifetime, particularly in the case of children, provided their causes cease to exist. The pulmonary tissue of children, although delicate and very yielding, is, on account of its great elasticity, peculiarly fitted for repair; but a long continuance of the affection gives rise to a genuine emphysema, *i.e.*, a rarefaction of the tissue, and the *restitutio ad integrum* then becomes impossible. We may say, then, in general terms, that an emphysema once established is *incurable*, although, from what we have already seen, it is of variable portent to the individual affected. An improvement, at least for a length of time, or, in moderate degrees of the disease, a certain arrest of its progress may, too, unquestionably be attained by a careful avoidance of all influences that are deleterious to the respiratory organs. *Death* may result from the most diverse accidents or intercurrent diseases, such as severe febrile bronchitis, catarrhal pneumonia, or an access of croupous pneumonia, from cardiac complications, apoplexy, etc., or from the results of the emphy-

sema, such as fatty degeneration of the heart, marasmus, and general dropsy. Cases have been observed, too, although not often, in which death has taken place by asphyxia during an asthmatic attack.

Interlobular and subpleural emphysema may end in recovery. More rarely it leads to the development of a pneumothorax, which threatens the patient's life. Excessive generalization of the emphysema, and its extension to the mediastinum and to the subcutaneous tissue of the neck and chest, may often prove fatal within a few minutes by compression of the heart and great blood vessels.

The *prognosis* in *substantive emphysema* is, according to present data, unfavorable as regards future health, whilst, as regards life, it is comparatively favorable, since, although indeed it shortens life, yet it may do so only after a long time. In individual cases the prognosis is to be founded upon a consideration of the following points: (1) The degree of development of the emphysema; (2) the accompanying catarrh; (3) the patient's age; (4) his constitution, the care which is taken of him, and his diet; and (5) above all, the condition and functional capability of his heart. The lighter grades of the disease may, then, often be borne for a long time, especially if the accompanying catarrh be moderate and occasioned only by particular noxious circumstances, or if it make its appearance during the cold season, to disappear again in warm weather and under the influence of good nursing. The severer forms of emphysema, accompanied with excessive dyspnœa and orthopnœa, and a prolonged and harassing dry bronchial catarrh, are not only a burden to the patient, but they also hasten the fatal issue. Advanced age, previous marked debility, a hydræmic condition, the lack of efficient nursing, unfavorable surroundings—such as unfortunately exist amongst the poorer classes—but above all, a failure of the heart's impulsive power, denoting a weakening and incipient degeneration of its tissue, first revealed by a trifling and scarcely noticeable œdema, which gradually advances to general dropsy—these circumstances render the prognosis in the highest degree unfavorable.

In *vicarious emphysema*, if it be not so excessive as to

become of intrinsic importance, like substantive emphysema. the prognosis will rest mainly upon the fundamental disease.

Diagnosis.

The diagnosis between a *substantive* and a tolerably diffused *vicarious emphysema* is not always easy, but yet the history of the case will furnish certain points which should not be underrated. If the dyspnœa and other symptoms of emphysema were not preceded by such diseases of the lung-parenchyma and bronchi as have been mentioned above under the head of etiology, or if they run their course without excessive expiratory paroxysms, and exist independently of any vocation or other circumstance, such as heredity, etc., favoring the occurrence of substantive emphysema, we should conclude that the case is one of vicarious emphysema.[1] Moreover, according to Niemeyer, an expiratory position of the thorax comports rather with a vicarious, and a barrel-shaped thorax with a substantive emphysema.

Moderate degrees of emphysema furnish no special characteristic symptoms, and often are only discovered incidentally in the dead body. In general, the recognition of emphysema rests upon the following data: the history and course of the affection; the existence of catarrh, dyspnœa, and asthma; the form of the chest and the type of respiration; enlarged pulmonary boundaries, with the liver occupying a low position; a diminished area of cardiac dulness, with epigastric pulsation and a lack of the apex beat; weakness or absence of vesicular breathing, with the fremitus preserved and the percussion note loud and sonorous; an exaggerated second pulmonic heart-sound; and signs of stasis, such as cyanosis and dropsy. It is indeed true that, even in case of a tolerably well-marked pulmonary emphysema, some important symptoms may be absent, especially when the disease is vicariously associated with wasting and destruction of the upper lobe of the lung, due to chronic

[1] The case cited by me above (p. 358) was undoubtedly one of substantive emphysema, since the pneumonia was cured without the formation of emphysema, which only occurred on the patient's resuming his occupation as a trumpeter.

pneumonia and tuberculosis. In such a case the sterno-vertebral diameter may even be diminished in the upper region of the chest, and the supra-clavicular fossa deeply sunken. But, even under these circumstances, such dilatation of the lower portion of the thorax as may happen to be present, the depressed position and impaired mobility of the diaphragm, and especially the diminished area of cardiac dulness, will render the diagnosis certain.

First of all, true emphysema may be confounded with a *simple dilatation*, the "permanent inspiratory distention of the pulmonary alveoli" of Niemeyer (following bronchitis and spasmodic asthma). In this case a decision can be arrived at only by further observation of the course of the affection, as to whether the lung returns to its former dimensions on the subsidence of the bronchitis or of the asthmatic bronchial spasm, or remains distended.

I have recently had under treatment a woman about thirty years of age, who was the subject of severe asthmatic attacks. During an access of this sort the boundary of the lung was extended downwards to the extent of about one and a half intercostal spaces, and the cardiac dulness and impulse were wholly wanting. Over nearly the whole surface of the lung very labored and prolonged expiration was to be heard, with marked sibilant sounds, whilst in certain regions the respiratory sound was well-nigh inaudible. Understanding that the woman had had a cough for a long time previous, and not having seen her before, I was inclined, at the first glance, to look upon the case as one of advanced substantive emphysema. When I saw the patient again on the next day, the asthma having been assuaged by hypodermic injections of morphine, the lower border of the lung had assumed a higher level, the area of cardiac dulness, although small, was present, and the apex beat was perceptible. In subsequent frequent repetitions of the asthmatic attacks, I was able in each instance to ascertain the same phenomenon.

In the diagnosis of emphysema from *pneumothorax*, the following points may be appealed to: Pneumothorax is always acute in its onset, and is unilateral, the thoracic walls are excessively distended, and the intercostal spaces prominent and immovable. The heart, together with the mediastinum, is pressed over towards the opposite side. The vocal fremitus is diminished, and the percussion note is generally tympanitic, and, if the pneumothorax be of long standing, varies with each change in the patient's position, on account of the supervention

of fluid effusion (pyopneumothorax). Moreover, in pneumothorax we find the well-known succussion phenomena, and, on auscultation, we generally hear amphoric breathing, metallic tinkling, and an amphoric vocal resonance.

Riegel[1] has published a case of emphysema of acute formation, which could be distinguished from a circumscribed pneumothorax only by the absence of metallic sounds. The patient was a man thirty-four years of age, who was suffering from infiltration of the summits of both lungs, and was suddenly attacked with severe dyspnœa and pain in the left half of the thorax. The lower portion of the left side of the chest was enlarged, the left intercostal spaces, from the fourth down, were even, the apex of the heart was pushed to the left border of the sternum, there was tympanitic percussion on the left side, and the respiratory murmur was rendered inaudible by non-sonorous râles, being vesicular only over small areas. At the autopsy, besides phthisis, the lower part of the left lung was found to be aerated, and at its very base there was a collection of sacs varying in size from that of a cherry-stone to that of a pigeon's egg.

The diagnosis of emphysema, when accompanied by a systolic mitral murmur, from heart disease, particularly *mitral insufficiency*, in which hypertrophy of the right side of the heart, bronchitis, and the most multiform symptoms of venous stasis are present, offers no special difficulties. In these cases of heart trouble, the peculiar shape of the thorax and the enlarged pulmonary boundaries are wanting. Instead of the area of cardiac dulness being curtailed or absent, it is enlarged, and, instead of the apex beat being imperceptible, it is visibly and palpably strengthened or diffused. In case of marked dropsy, or of emphysema complicated with mitral insufficiency, as well as of kyphosis and other deformities of the thorax, the diagnosis may be more difficult.

Only in very rare instances, surely, would an *aneurism of the arch of the aorta* or of the *subclavian artery* be confounded with emphysema ; an example of this kind, however, is given by Biermer. This might happen in case of dyspnœa and of dilatation of the lungs from secondary bronchial catarrh, especially if dropsical symptoms were associated with enlargement of the heart, and if, as is not infrequently the case, there were absence of laryngeal and œsophageal symptoms, and of the pulsations

[1] Bayr. ärztl. Intelligenzblatt, 1872, 16. Virchow-Hirsch, Jahresber., II., 118.

and characteristic aneurismal sounds, due to filling up of the sac. We can here guard against error only by a thorough examination of the vascular system. (Compare the section on Aneurisms.)

Interlobular and *subpleural* emphysema can be diagnosticated only when it has extended into the mediastinum and to the subcutaneous tissue.

Treatment of Emphysema.

The *indicatio morbi* will not suffice for our guidance in this respect, since, when once the lung tissue has become inelastic and atrophied, we are unable to restore it to its pristine normal condition. Therefore the emetics and tonics, which have been recommended for this purpose, are of no effect. The pressure which attends retching and vomiting would, it has been supposed, squeeze out the pulmonary alveoli, and thus gradually reduce their size. But we know that emphysema affects precisely those parts which are scarcely, if at all, subjected to this pressure, namely, the apices of the lungs. Emetics may be indicated under some circumstances, but on quite different therapeutic grounds. The same is true in regard to the use of tonics and motor-excitants, such as nux vomica and strychnine, which, according to the recommendation of Stokes and Martin, have been considered to increase the tone of the bronchi and pulmonary vesicles and contract the relaxed lung tissue.

For the same purpose Koch[1] has recommended arsenite of antimony (in the form of pills, each containing one thirty-second of a grain of this substance, with one thirteenth of a grain of muriate of morphia, of which one is to be taken daily at first, increasing subsequently to as many as six daily) as a nerve and muscle tonic capable of correcting the weakened contractility of the lung tissue.

The *causal* or *prophylactic indication* involves the treatment of the original disease in cases of vicarious emphysema. As regards substantive emphysema, we must, above all else, allay the severe fits of coughing incident to intercurrent diseases, such as bronchitis and whooping-cough, by such appropriate treat-

[1] Presse Méd. Belge, 1870, 5.

ment as will be found described in the sections devoted to those affections. It is further to be remarked, that extensive inflammatory affections of the lung substance, even after the disease proper has been subdued, and no vestiges of it are, at least on physical exploration, longer to be observed, may, under certain circumstances, predispose to the formation of emphysema. On this account, such patients should wait a long time—how long is uncertain, and varies greatly in individual cases—before resuming their occupations, provided these involve forcible expiration (as in playing upon wind instruments, glass-blowing, lifting heavy weights, or severe bodily exertions).

The *symptomatic indication* covers most that remains for us to do. *Bronchial catarrh*, in particular, first claims our attention, for not only may it be the primary causative factor, but in many cases also it is originated and kept up by the emphysema, and, when once present, it contributes essentially to the aggravation and spread of the emphysema. It should be treated dietetically, as well as medicinally, according to the rules laid down in previous chapters.

In the case of young and vigorous subjects affected with bronchial catarrh and incipient emphysema, we may seek, by means of cold baths and douches at any time of the year, and also by means of sea-bathing, to render them less sensitive to changes of temperature. If the emphysema be of long standing, and if the patient be somewhat advanced in years or very sensitive, we should let him keep his room in raw weather, during easterly and northerly winds, and in the cold seasons—the latter part of autumn, winter, and early spring; or, if his pecuniary circumstances admit of it, we may let him reside in a southern climate during such seasons. Moreover, he should take care always to keep his feet warm. Summer and winter he should wear flannel over the whole trunk, perhaps exchanging it for shirting or silk during the summer and in warm districts.

As regards treatment by means of drugs, it is recommended, in case the cough be dry and very urgent, so as to interfere with rest at night, that a dose of morphine or Dover's powder be taken in the evening, before going to bed. The alkaline and saline muriatic springs are also to be recommended. It is better

to take these waters at the localities where they are naturally found; but, if this be impracticable, the water may be drunk at home, before breakfast, with equal parts of warm milk. On the other hand, the saline vapors are too irritating in cases of dry catarrh, and are appropriate only to those accompanied by hypersecretion. Inhalations of alkalies, such as bicarbonate of soda, or, if there be excessive secretion of mucus, of balsamics, oil of turpentine, tar-water, etc., produce tolerably good results. Warm baths and vapor baths, which have a diaphoretic effect, should be cautiously employed, and, indeed, only when the disturbances of the circulation and respiration are not yet of importance, and the compensatory action of the heart is unimpaired. For the rest, we may refer to the chapter on Bronchial Catarrh.

Further treatment is demanded by the *dyspnœa*, so annoying to patients, which is the direct result of the carbonic acid poisoning, and which may even amount to orthopnœa and asthmatic attacks. Inordinate bodily exertion and fatiguing walks should be strictly forbidden, whilst moderate exercise is very much to be commended. Regular and sufficient defecation should be procured, and all flatulent, acescent, and fatty articles of food, as well as late suppers, should be avoided. It is best that the patient should have a simple, miscellaneous diet, and content himself with a dish of milk-broth and a little white bread in the evening. If there be accumulations of mucus in the bronchi, and the dyspnœa be due to inability to dislodge them, active expectorants should be used, and, in asthmatic seizures, emetics and such excitants as benzoin, camphor, and musk. The narcotics—opium, morphine, cherry-laurel water, belladonna, hydrate of chloral, etc.—are of use only when there is actual spasm of the muscular tissue of the bronchi. Under such circumstances also nauseants are indicated, such as a weak infusion of ipecacuanha and tartar emetic. Many use iodide of potassium, in doses of from one and a half to four and a half grains in pill or solution. Derivation to the skin, by means of sinapisms applied to the chest and the feet, and friction of the legs with spirit of mustard or camphor, is also indicated. In addition, a sojourn in pine woods, a sea voyage to mild climates, and the inhalation of compressed air in pneumatic institutes and cabinets

(like those of Lange in Dresden, Marck in Reichenhall, and others) are recommended.

According to Tabarie, Pravaz, Bertin, Vivenot, and Lange, the latter is a very efficient curative measure. According to Bertin, out of ninety-two cases treated with baths of compressed air, sixty-seven would seem to have been perfectly and permanently cured. The catarrh, in particular, was either cured or very much ameliorated, and the patients were rendered more capable of resisting all the exciting causes of catarrh. In case catarrh actually made its appearance, dyspnœa and oppression were absent, or at least very moderate. The efficiency of this method of treating emphysema is supported, according to the most varied observations, by the calmative effect of the compressed air upon the respiratory and circulatory organs in health and disease, and in its retarding the breathing and the heart's action, so that the heart contracts more perfectly and satisfactorily, and becomes more capable of thoroughly emptying itself of blood; and, moreover, as observed by Vivenot, by the increase of the vital breathing capacity, and by the augmented exhalation of carbonic acid and admission—*i.e.*, absorption—of oxygen. Finally, by facilitating the respiratory function, compressed air effects an increase of tissue-metamorphosis, a better appetite, an improved nutrition, and therefore an augmentation of strength and bodily weight. According to Biermer, the effect of compressed air is further shown (1) in that the air pressure, heightened by one and a quarter to one and a half atmospheres, promotes the expiratory design of the elasticity of the thorax and of the intestinal gases; (2) in that, by the increased pressure upon the vessels lying without the thorax, the return of the venous blood is facilitated, and, by an analogous action upon the mucous membranes, the catarrhal hyperæmia of the bronchi is lessened; and (3) in that, by the relatively high oxygenation of the air entering the lungs, the *besoin de respirer* felt by the emphysematic is mitigated.

Moreover, it has been ascertained, by means of Waldenburg's method of *pneumatometry*,[1] and Riegel's *stethography*,[2] that a

[1] Berliner klinische Wochenschrift, 1871, No. 45.

[2] Die Athembewegungen. Würzburg, 1873.

dyspnœa may depend either upon insufficient inspiration, upon insufficient expiration, or upon both together. It is clear, from previous demonstration, that in emphysema it is really the expiration which is insufficient, and, according to Waldenburg, the inspiratory act either remains normal, or, on account of the excessive use made of the inspiratory muscles, which have acquired increased power, may even exceed the norm. This suggested the idea of facilitating the expiratory act in emphysematics, by sucking out, as it were, the air which the inadequate expiration fails to expel, the patient being made to breathe out into rarefied air. An apparatus was constructed for this purpose by J. Hauke,[1] and modified by Berkart,[2] into which, by means of a pair of bellows, air could be pumped, or withdrawn from it, and from which air, either rarefied or compressed, could be conducted to the patient through a pipe. According to Waldenburg, however, this apparatus has two important defects. Thus, it admits of only a very slight development of force, and it works irregularly, the maximum of air pressure gradually sinking to the minimum, unless fresh air be pumped in, or undergoing continual fluctuations, if the pumping be kept up. These defects are avoided in the apparatus described and used by Waldenburg.[3] By means of this the compression or rarefaction of the air may be carried to any desired extent, and its working force is perfectly uniform. As regards the construction and mode of use of this apparatus, we must refer to the original publication.[4]

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[1] Ein Apparat zur künstlichen Respiration, etc. Wien. 1870; and Appendix to the brochure: Ein Apparat für künstliche Respiration. Wien. 1872.

[2] Lancet, 1871, No. 25.

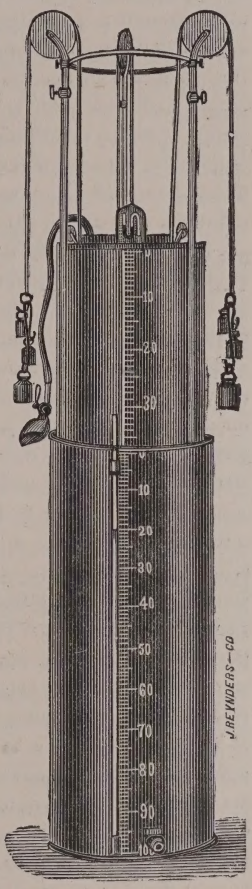
[3] Berl. klin. Wochenschrift, 1873, Nos. 39 and 40.

[4] As the article here referred to is probably not accessible to a majority of English and American physicians, I have no doubt the following abstract of it, very kindly prepared by Dr. J. Haven Emerson, of New York, will prove an acceptable addition to the work.—EDITOR'S NOTE.

In the treatment of pulmonary diseases the attempt has been made in various ways to obtain the advantages of different degrees of density in the air breathed. Patients have been sent to elevated (Alpine) regions; they have been shut up in an air-tight cabinet, the air of which could be condensed or rarefied at pleasure, and finally, in 1870, Hauke invented his apparatus, by which patients were enabled to breathe from or into either condensed or rarefied air, through the medium of a mask connected with a

In the employment of this apparatus, which is remarkable for its simplicity, handiness, and portability, it is evident that, in emphysema, the quantity of air expired into the cylinder (on

receiver. Waldenburg's attention was directed to this subject, and, in connection with it, he and Riegel developed the knowledge of the respiratory movements and a system of pneumatometry. Waldenburg analyzed the respiratory act, and determined, with regard to different pulmonary diseases, whether the dyspnœa associated with them was due to a defect in inspiration or expiration. Thus he considers it as established that in emphysema the expiratory act only is insufficient. In phthisis, on the other hand, he finds the inspiration primarily and chiefly deficient, although the expiration suffers at a later stage of the disease. Associated with stenosis of the upper air-passages, inspiration only is at fault. After some experimentation with Hauke's apparatus, he satisfied himself that there were several minor defects in it, and two important ones, viz., the limited degree of condensation or rarefaction that could be obtained by it—not more than about $\frac{1}{40}$ of an atmosphere—and the unevenness or inequality of its working. With the object of remedying these deficiencies he devised the apparatus figured below.



It is made of sheet zinc, and consists of two cylinders, the larger 1 metre long and 30 cm. in diameter, in which water is put, and the height at which it stands indicated by means of a glass tube which runs up on the outside and is graduated to centimetres. This cylinder has also a stopcock on a tube at the bottom. The smaller one is 27 cm. in diameter, and fits into the open top of the larger cylinder. Its bottom is open, and its closed top has two orifices in it, to one of which is fitted a manometer, and to the other a flexible tube, also supplied with a stopcock, and ending in the mask used in inhaling. Three rods rise from the sides of the outer cylinder, which are furnished with wooden wheels at their tops, over which play the chains which support the inner cylinder, these being fastened to the top of the latter. The free extremities of the chains which hang down outside have cross-bars or hooks, to which weights can be attached, the whole arrangement being very similar to the large receivers at the gas-works. The effect of raising the inner cylinder by hanging weights on the chains, is to rarefy the air it contains, while, when the stop-cock at the top is opened and the cylinder is filled with atmospheric air—which can readily be done by simply drawing up the smaller cylinder—this result of sinking it again by placing weights on the top will be to condense this air. The weights added to increase or diminish the density of the contained air are ordinarily from 20 pounds, representing a pressure of $\frac{1}{65}$ atmosphere, to 40

account of the rarefaction of the air in the apparatus) is greater than the vital capacity of the lungs, as measured by the spirometer. The excess must therefore come from the *residual air*, and, by reason of the alternation of deep inspirations and expirations, a very extensive ventilation of the lungs must take

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pounds, or $\frac{1}{20}$ atmosphere, and Waldenburg doubts if it is advisable to exceed 60 pounds, or $\frac{1}{15}$ atmosphere.

Such being the apparatus, it is available for several uses, depending on whether the air contained in it be condensed or rarefied, and whether the patient practise inspiration from it or expiration into it. The two principal ways in which it has been used are the following: *First*, the air in the receiver being condensed, the patient draws this condensed air into his lungs in inspiration. He holds the mask firmly against his mouth and nose with the left hand, and controls the stopcock with the right. He should open the mouth and breathe deeply and steadily. The number of respirations required to exhaust, and so depress the cylinder, varies according to the capacity of the lungs of the individual, and serves as a guide to the length of time that the apparatus should be used at one sitting. It should be noticed that the principles of construction are such that the air in the receiver remains of uniform density until it is exhausted. Usually a sitting of from ten to twenty minutes, at which the receiver is emptied from one to three times, is sufficient. Persons whose lungs are of small capacity, *e.g.* women, or those suffering from emphysema, may require from twenty to thirty breaths to empty a receiver which others would empty in from five to eight. Thus the capacity increases with practice, and is an index of improvement. The condensed air then pours into the lungs, and, until accustomed to it, the patient, or indeed a healthy person, has a slight sense of oppression. Soon, however, he finds that his breathing is easier than usual, and his chest becomes distended. And this is not merely a subjective sensation, but an actual fact, as proved by the spirometer. The interchange of gases in the lungs is accelerated, and dyspnœa diminished or removed. It is also found that the ultimate effect is an increase of inspiratory force. The *second* method may be used by itself or combined with the one just described. It consists in breathing out into the receiver when it contains a rarefied air. The process and rules to be observed are wholly analogous to those just described in regard to inhaling the condensed air. Of course the effect is to empty the lungs more thoroughly than usual, at the expense of their residual air, and this being naturally followed by a deep inspiration, the effect is a thorough ventilation of the lungs. There is at first a sensation of compression of the chest, notably of the lower ribs and lower part of the sternum. The relief and permanent advantages gained are similar to those attained by the method first described, and in emphysema it has been ascertained by percussion that the lungs, which had extended beyond the lower border of the ribs, and advanced over the præcordial space, became retracted to their normal dimensions, thus indicating a positively curative action upon the disease by a purely mechanical agency. Expiration into rarefied air produces the most marked increase of the expiratory force. The *third* mode of using the apparatus, viz., by the inspiration of rarefied air, has been but little employed. Still, Waldenburg regards it as a powerful gymnastic exercise for the lungs, and likely to be useful in developing the

place. That the lungs are thereby more than ordinarily retracted, and that the space for the residual air is diminished, is proved by clinical observations, in which there is noticed not only an increase in the amount of air expelled from the lungs, but also an exaltation of the respiratory forces, even up to the normal standard. Thus the previous expiratory insufficiency is mitigated or wholly overcome. It may even be ascertained, by means of percussion in these cases, that the lungs have returned to their natural dimensions.

Inspiration of air compressed to a sufficient degree is capable of converting the negative into a positive pressure. In this manner the afflux of blood into the aortic system will be increased, and its efflux from the veins into the right side of the heart diminished, thus augmenting the amount of blood in the systemic circulation, and, *ipso facto*, lessening that in the pulmonary circulation, *i.e.*, in the lungs. On the other hand, expiration into rarefied air will cause the blood, by reason of decreased air pressure within the lungs, to be more forcibly drawn into the right side of the heart and into the lungs, whereby the latter will become highly overcharged with blood. Now, in emphysema with marked bronchial catarrh we find the lungs hyperæmic, and, therefore, by making use of rarefied air we should only still further increase the hyperæmia. Hence it is necessary under these circumstances to combine, in emphysema, the inspiration of compressed air with expiration into attenuated air. Waldenburg is in the habit of allowing compressed air to be inspired first for a period of from five to fifteen minutes, and then, after a pause of equal duration, expiration into rarefied air to be performed; and in many cases he allows another final inhalation of compressed air. According to the publications of Waldenburg, Sommerbrodt, and others, the very favorable

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chest in persons of phthisical tendencies or in the early stages of that disease. It may be used in combination with the inhalation of compressed air.

Quite recently there has been introduced by Dr. Ph. Biedert a new form of apparatus, in which the principle of the bellows is made use of. It is described by Dr. A. Rose in The Medical Record of Aug. 28, 1875. While it offers some advantages over the present form of Waldenburg's apparatus, it seems probable that better results may ultimately be obtained from the use of water pressure as employed in the latter.

results attained by this method entitle it to a place in the recognized treatment of emphysema. It is contraindicated in cases where there is secondary degeneration of the heart.

With the same end in view, to wit, assistance of the expiratory act, *compression of the thorax* has also been recommended, to restore the chest-walls and the diaphragm from their permanent inspiratory to their normal position. In order, however, to avoid undue straining of the muscles of the arms, the compression should not be carried out by the patient himself. Gerhardt has recently in two instances obtained a good result by employing this old method, which has been placed upon a new and firm basis by the stethographic and pneumatometric investigations of F. Riegel and Waldenburg. As was to be expected, the younger the patients and the more pliable and yielding their costal cartilages, the better the results. According to Gerhardt, this method of treatment is most appropriate in cases in which the muscles are too weak to overcome the obstruction of the air-passages by thickened secretion. The assistance in these cases is direct by means of the expiratory pressure, and indirect by means of the strengthening of the muscles resulting from the easier absorption of oxygen. After the daily use of compression for several weeks, an increase of the vital capacity was shown, a less laborious respiration, with diminution of the subjective dyspnœa, promotion of the expectoration, elevation of the diaphragm, and, by reason of the reduced size of the lungs, a greater area of cardiac dulness. Under this treatment, however, two unpleasant effects were observed, namely, *pulmonary hemorrhages* and *convulsions*. Gerhardt is of the opinion that the latter were due to carbonic acid being driven from the obstructed air-spaces into the blood by the pressure employed, thus occasioning an increased accumulation of carbonic acid.

Faradization of the phrenic nerves and of the abdominal muscles is of no use in the treatment of the dyspnœa.

Asthmatic attacks, as already stated, are generally overcome by narcotics, and, in case they show a certain type, quinine in large doses, with or without morphine, is indicated. In addition, iodide of potassium, bromide of potassium, bromide of ammonium, oxide of zinc, valerianate of zinc, various anti-spas-

modics, arsenic, and lobelia are employed, but with no marked effect.

Cyanosis and *dropsy* call for special treatment. Venesection is seldom indicated in these cases, since it would only make the blood still thinner, and increase the dropsy. It is in just this stage of defective compensation, too, that any lowering of the powers by loss of blood would prove of positive detriment to the heart's action. In cases of marked over-distention of the right side of the heart, general bloodletting, with the simultaneous use of stimulants, may be indicated.

In the early course of dropsical symptoms they will generally yield to a diaphoretic treatment and the employment of drastic purgatives. At a later period, if the œdema become more marked, these measures are no longer of service, and we must resort to digitalis either in infusion or in pill form, in doses of from one-half to one grain from four to six times daily. On a still further increase, even this is of no avail, and we must then have recourse to the other diuretics. I generally use cream of tartar at first, and afterwards acetate of potassa with oxymel of squill or extract of juniper, or (as advised by Niemeyer) three and a half drachms of vinegar of squill, together with as much carbonate of potassa as can be dissolved in five ounces of water. Oppolzer recommends, also, the decoction of ononis spinosa, the infusion of uva ursi leaves, or the infusion of juniper berries in combination with a solution of acetate of potash containing four scruples of the salt and half an ounce of oxymel of squill.

I can recommend the boro-tartrate of potassa as the best remedy, which will often reduce to a minimum the very marked dropsical swellings of the limbs, the scrotum, and the penis, and even collections of water in the abdominal cavity. (℞ "Tartari boraxati" ʒ ss., oxymellis scillæ ʒ ss., aquæ destillatæ, vel aquæ petroselini[1], f ʒ v. M. S. A tablespoonful every second hour.) But unfortunately, on account of the insufficiency of blood in the aorta, these remedies always cease to promote the urinary secretion after a certain length of time. The dropsical phenom-

[1] Parsley seed, 1 part; water, a sufficient quantity; distill off 20 parts.—*German Ph.*

ena reappear and become extremely widespread on the approach of death.

In extreme cases of emphysema it is recommended to assist the heart's action by *stimulants*, such as the stronger wines (port, Madeira, and champagne), anisated spirit of ammonia,[1] benzoin, camphor, and musk. I have seen good results from the use of carbonate of ammonia in doses of from one-half to two and a quarter grains every second hour, in wafers.

As a matter of course, the accompanying digestive derangements should receive appropriate treatment, and, in cases of gradual loss of strength, *roborants* should be used, particularly iron and quinine. The lactate of iron is very well borne by patients, and likewise the reduced iron made into pills with extract of cinchona.

Interlobular and *subpleural* emphysema is seldom to be recognized, and accordingly is not amenable to treatment. Only when it extends into the mediastinum, giving rise to severe dyspnœa and oppression, is the use of narcotics (morphine, opium, belladonna, and cherry-laurel water) indicated. Moderate subcutaneous emphysema may, in the absence of other urgent symptoms, be treated on the expectant plan, or, as Roger suggests, we may endeavor to favor absorption by means of dry and moderately irritant frictions. In the more marked cases of this sort, large scarifications should be made in the skin, according to the rules of surgery. In cases of excessive cyanosis, with impending suffocation, venesection may be employed.

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[1] Oil of anise, 1 part; dissolve in alcohol, 24 parts; add water of ammonia, 5 parts.—*German Ph.*

GANGRENE OF THE LUNG.

SPHACELUS, MORTIFICATIO, GANGRAENA PULMONUM.

HISTORY AND LITERATURE.

The first accurate knowledge and description of gangrene of the lung came from *Laënnec* (Traité de l'auscultation), who demonstrated both the forms which are still commonly recognized. Next come the important works of *Cruveilhier* (Anat. pathol., Livr. III. et XI.), who considered occlusion of the vessels to be the cause, and described dry gangrene; also the works of *Schroeder van der Kolk* (Observations anat. path., I., 202), where we find a good anatomical description of diffuse gangrene. *Corbin* (Journ. hebd. A., VII., 126) distinguishes superficial gangrene from other forms. *Andral* (Cliniq. méd. Ed., IV., tom. III.) and *Carswell* (Elementary Forms of Disease, Mortification, Pl. III.) describe cases of gangrene following inflammation of the lungs. The latter author explains the death of a portion of a pneumonic lung as depending upon the interference caused to the circulation by the lateral pressure exercised by the infiltrated tissue upon the vessels.

Guislain (Gazette méd., IV., 33) describes gangrene occurring in the insane from unwillingness to take food; *Genest* (Gaz. méd., IV., pp. 593 and 657) gangrene following pulmonary apoplexy.—*Tournet* (Expérience, I., 322).—*Stokes*, Diseases of the Chest.—*Gerhardt* (Annal. méd. Belg. étrangers, 1838, Aug. and Sept.) records from the Hospital of Philadelphia a great number of cases which force him to adopt the opinion that gangrene occurs most commonly as a primary and idiopathic affection, in individuals who are reduced in vigor by the abuse of alcohol, poverty, or such like lowering circumstances.—*Laurence*, Expérience, V., pp. 321 and 337.—*Craigie*, Edin. Med. and Chir. Rev., 56, 1841.—*Briquet* (Arch. gén., CXI., 5) describes gangrene from bronchiectasis.—*Grisolle*, Traité de la pneumonie, p. 346.—*Rilliet* and *Barthez* (Maladies des enfans, CCVII.) give some important communications concerning pulmonary gangrene in children.—Also *Roudet*, Arch. gén. D., II., 285, and III., 54.—*Mosing*, Oesterr. Jahrb., 1844, April and May.—*Durrant*, Dublin Med. Press, Nov., 1846.—*Lieblein*, Heidelb. med. Annal., 1846, XII., 1.—*Fischel*, Prag. Vierteljahrschrift, 1847, IV., 1.—*Virchow's* works on Thrombosis, Embolism, and Metastasis (gesammelten Abhandlungen. Frankfurt a. M., 1856).—*Rapp*,

Ueber Bronchektasie, Verhandlung d. Würzb. med. Gesellsch.—*Dittrich*, Ueber Lungenbrand in Folge von Bronchialerweiterung. Erlangen, 1850.—*Stokes*, Dubl. Journ. Med. Sci., 1850.—*Scoda*, Wien. Wochensch., 1852, 15.—Through the numerous classical works of *Traube* (Deutsche Klin., 1853, 37; 1859, 46; 1861, 50 et seq., as also his gesammelte Beiträge zur Path. und Therap.) not only was our knowledge in diagnosis placed upon a sure basis, but it also became possible to distinguish between gangrene and putrid bronchitis; while *Scoda* created a new era in the therapeutics of the affection, especially the local treatment.—*Virchow*, Würzb. Verhandl., 1851, II., 2.—*Lorrenzis*, Nonnullæ de pathologia gangrænæ pulmonum. Kiel, 1854.—*Weinberger*, Oesterr. Zeitsch. für prakt. Heilk., 1855, I., 45, 46.—Revue clinique hebd. de quelques cas de gangrène pulmonaire; diagnostic et traitement.—Gaz. des Hôp., 108, 1856.—*Henchel*, De la gangrène du poumon. Thèse. Strassb., 1856.—*Troussot* (Union méd., 1857, Nrs. 67, 68, 69).—Gazette des Hôp., 1857, 109, 111, 115.—*Kaulich*, Ueber d. Lungenbrand. Prag. Vierteljahrsch., 1861, I.—Bericht aus der Klinik von Prof. Jaksch, von 1857 bis 1859.—*Gamgee*, Edin. Med. Jour., 1865, March, with an addition from Laycock; ib. May.—*Jaffé*, Lungenbrand durch einen verschluckten Kirschkern erzeugt. (Med. Centralzeitung, 1866, No. 27).—*Leyden* und *Jaffé*. Ueber putride (foetide) Sputa, etc. Deutsches Arch. fur klin. Med., II., p. 488, and Berl. klin. Wochenschr., 1867, No. 1.—*Scoda*, Zur Path. u. Ther. d. Lungengangrän. Wien. med. Presse, 1867, 4.—*Banks*, Clinical Reports and Observations. Dubl. Jourl., 1867, Feb.—*Hertz*, Arch. f. path. Anat., Bd. 40, 3 and 4.—*Leyden*, Ueber Lungenbrand. Volkmann's Vorträge, 26.—*Lebert*, Klinik d. Brustkrankheiten. Tübingen, 1874, I., p. 802.—Compare also the various handbooks of Pathological Anatomy.

ETIOLOGY.

GANGRENE of the lung consists in death of the lung tissue, associated with putrefaction owing to the admission of air. In this it is distinguished from necrosis of other tissues, where communication with the atmosphere is shut off. Gangrene of the lung is a comparatively rare affection, which, according to Laënnec and others, appears to occur more frequently in men than in women. The cases published by Kaulich support this view, as of the nine cases which occurred in the clinique of Jaksch, seven were men and two women. Lebert also found twenty-two men among thirty-two patients with this disease. With reference to the *age* of the persons attacked, it is at least certain that the years about puberty and the middle period of life show the greatest number of cases.

According to the statistics of Laurence, gangrene of the lung occurred once between the ages of 1 and 10; five times between 10 and 20; seventeen between 20 and 30; twelve between 30 and 40; fourteen between 40 and 50; ten between 50 and 60; and from 60 to 80 there were four cases recorded. Out of thirty-two cases, Lebert found none between the ages 1 and 10 or 65 and 70, while there appeared between 16 and 20, 46 and 50, 56 and 60, 61 and 65, one case each; between 11 and 15, two cases; between 31 and 35 three cases; between 26 and 30, 36 and 40, 51 and 55, four cases each; and between 21 and 25, seven cases.

The disease may appear as an *idiopathic* or primary affection, though more commonly it develops itself *consecutively* during the progress of some other lung disease, or it may be of *metastatic* nature. Its origin may generally be traced to some of the following causes:

1. *Interruption*, or *weakness of the blood-current*, in the lung. 2. *Decomposition* by means of chemical agents. 3. *Lowering of the general nutrition.* 4. *Traumatic* influences. Any one of these causes suffices for the production of gangrene of the lung; several of them, however, are often found to take an active share in the production of the same case.

One of the most frequent causes, and, indeed, the only cause which was recognized before Laënnec's time, is croupous pneumonia; particularly when it attacks impoverished, decrepid, or old people, or drunkards or individuals who have been previously weakened by acute or chronic diseases, such as measles, small-pox, typhus, cancer, diabetes, parturition, etc. In this case the hindrance to the circulation depends upon the pressure exerted on the capillaries of the lung by the inflammatory exudation. A defective condition of the blood must also be taken into consideration. Cases are also known where gangrene sets in at the very height of an attack of acute pneumonia in plethoric young persons. In these cases hemorrhage during the exudation stage may be looked upon as the inducing cause, as Rindfleisch states.[1]

Lately a butcher, æt. 32, who had been previously healthy, came under my care. Five weeks before he had been attacked with rigors, stitch in the side, and cough coming on after a sudden chill. The next day a copious discharge, almost entirely composed of blood, was expectorated: the sputa only acquiring the tenacity and

[1] Lehrbuch, etc., 3. Auflage, p. 392.

rusty color characteristic of pneumonia several days afterwards. Fourteen days from the start of the disease the patient and his neighbors began to remark that his sputa and breath had an extremely disagreeable smell. As the treatment adopted did not remove this very unpleasant symptom, and the patient continued to be feverish, to sweat and lose flesh, he sought my aid. Both the macroscopic and the microscopic characters of the sputa, as also the evidence of a pneumonic infiltration in the lower lobe of the right lung, left no doubt in my mind as to the correctness of the diagnosis, "gangrene of the lung."

Interruption to the circulation in the lungs may also be produced by an *embolus*, which may either arise from a clot in the right heart, or owe its origin to some thrombus in the veins of the general circulation. The hemorrhagic infarction which results from this can produce putrefaction, destruction, and gangrene of the lung tissue, by causing complete stasis in the neighboring vessels. When both the agents—pneumonia and embolus—exist, the development of gangrene becomes still more probable, as occurred in the case published by me.[1]

To the *chemical influences* belong such emboli as have their origin in thrombi lying in *infective purulent centres.* Being tainted with the products of putridity, they give rise to a gangrenous inflammation in the part of the lung which is attacked. One finds such cases after amputations, bone suppuration, gangrenous bedsores, parturition, abscess of the liver, and caries of the temporal bone. In the latter case, as Traube[2] twice observed, clots adhering to the wall of the corresponding internal jugular vein are occasionally seen, as well as hemorrhagic infarctions of the lungs. Leyden suggests that the thrombi come from the veins of the petrous portion of the temporal bone; but this, however, can seldom be demonstrated. Volkmann[3] disputes this mode of origin of pulmonary gangrene, and believes that in these cases it depends rather upon the direct transmission of the carious pus through the Eustachian tube into the throat, and thence into the air-passages. Where neither venous thrombosis and emboli, nor infarctions in the lungs can be demonstrated, the question must remain unsettled; for it is

[1] Virchow's Arch., Bd., 40, p. 580.

[2] Deutsche Klinik, 1853, No. 37.

[3] Klinische Vorträge, No. 26, p. 206.

certain that gangrene may occur in the lungs by means of pus flowing into the smaller bronchi, as occurs in cases of purulent affections of the mouth and throat (in carcinoma of the tongue, jaw, tonsils, or pharynx, in gangrene of the lips after stings of insects, diphtheritis of the fauces, or operations on the cavity of the mouth and nose). Moreover, gangrene of the lung may be brought about, where *putrid suppuration* is going on *in the neighborhood* of the air-passages, by perforation of the ulcer into a bronchus, or into the lung, when adhesions have been formed with that organ. This may take place from suppuration in the thoracic or abdominal cavities, cancer of the œsophagus, caries of the vertebræ, retropharyngeal abscess, ulceration of the lymphatic glands, purulent pleuritis, perforating ulcer or cancer of the stomach, abscess of the liver, etc., etc. *Foreign bodies* which have found their way into the lung through the trachea, especially particles of food which rapidly decompose under the influence of heat, air, and moisture, may establish bronchopneumonia, accompanied by abscess or gangrene of the lung. This frequently occurs during the artificial feeding of lunatics or paralyzed persons, and in disease of the larynx or imperfect closure of the epiglottis. Other foreign bodies, which have little tendency to decompose, may set up gangrene of the lung in the same way. Thus Jaffé found an encrusted cherry-stone in the bronchus leading to a gangrenous centre, and Leyden a piece of bone as large as the joint of a finger in the midst of a gangrenous mass. The stagnant contents of the tubes in *putrid bronchitis*, or the matter contained in a *pre-existing cavity* of the lung, in a dilated bronchus (Dittrich), more rarely in a tuberculous cavity, and even in the sac of an echinococcus, may become decomposed, and by irritation of their boundaries may cause inflammation and ulceration, which induce gangrene and a kind of diffuse mortification of the neighboring lung tissue.

It is a doubtful question *whether lowering of the general nutrition* can by itself lead to gangrene, as has been described in drunkards, in persons suffering from diabetes or scurvy, in epileptics (?), in lunatics who refuse their food, and in persons suffering from inanition from such causes as stricture of the

œsophagus, hunger, want, bad air, or improper dwellings. It would rather appear that this condition produces an increased receptivity and want of power of resistance. Thus there may easily arise asthenic pneumonic infiltrations, which are often overlooked owing to the paucity of symptoms or the insufficient examination of the patient. Pneumonia in this class of persons often leads, as above stated, to gangrene, and the lung affection is not recognized until it betrays itself by the stinking sputa.

Leyden justly called attention to a mode of origin of gangrene of the lung which up to that time had escaped notice, namely, from *injury*. This occurs in case of lesions caused by stabs or gunshot wounds, when under unfavorable circumstances the opening does not heal, but by means of intense reactive inflammation, with considerable infiltration, the surrounding parts are thrown into suppuration, with the formation of abscess and gangrene. It may also be found to occur from severe contusions of the thorax, for instance, injuries to the shoulder. The elasticity of the chest-wall allows the contusion to act on the lung, and thus to produce effusion of blood, followed by gangrenous sloughs.

Whether gangrene of the lung occurs as an epidemic, as is reported of the prison of Lemberg by Mosing, appears doubtful, for the author's account of it leaves much to be wished for.

Pathological Anatomy.

Gangrene has been divided, since the time of Laënnec, into two forms—*circumscribed* and *diffused*. Both forms may occur in the same patient at the same time, and the first may pass into the second.

The *circumscribed* form of gangrene may be recognized, when it is situated near the periphery, by a sinking in of the lung tissue. When it reaches the pleura, the latter will be found covered with fresh fibrinous exudation. Upon section it appears first as a dark-brown or blackish, hard, dry slough, like that produced by a caustic alkali; this is commonly sharply defined from the neighboring tissue, which may be either simply œdema-

tous, hepatized, or infarcted. Sometimes the gangrenous piece commences to soften at the periphery, and to separate itself from the surrounding tissue at certain points, while at others it remains attached. Sometimes it separates completely and is cast off as a sequestrum, or it may become converted into a soft, diffluent, brown or black, stinking, putrid mass, with which are mixed the *débris* of tissue and dirty-white friable particles. The cavity thus formed is lined by soft polypoid shreds of sloughy tissue, and is traversed by such of the larger vessels and bronchi as still exist. The latter often open freely into the gangrenous cavity, and thus the contained fluid mass is allowed to escape.

The gangrenous spot may be situated in any part of either lung, but it is found more frequently at the periphery than in the central portion of the lung, and in the lower than in the upper lobe. According to Rilliet and Barthez, in children the right lung, and oftener the upper than the lower lobe, is the one usually attacked. From Lebert's compilation of his own cases and those of other observers, the frequency with which the right lung appears to suffer in comparison with the left is as 3 : 2. Sometimes both lobes are affected. The foci of disease may vary in *size* from that of a pea or bean to that of a closed fist, or sometimes they attain a greater size. Their *form* is round or irregular, their *number* single, or more commonly multiple.

The gangrenous centre seldom restricts itself to its original size, for it generally enlarges at its periphery, by the putrid mass setting up fresh centres of inflammation, which in their turn undergo decomposition. After the evacuation of its contents through the bronchi, the circumscribed gangrenous mass may undergo a kind of *imperfect healing process*, in which case the remaining shreddy sloughs are cast off from the walls of the cavity, which becomes lined with a tough, often hyperæmic membrane, composed of connective tissue, the result of a chronic process of reactive inflammation. This forms a kind of pyogenic membrane, and keeps up a continuous supply of pus. More rarely a *complete recovery* takes place ; but this appears to happen only when the centre is small and single. In this case the newly formed connective tissue contracts forcibly together, and

rich granulations spring up inside the walls of the cavity, which lie in such close apposition to one another that they finally grow together, and become converted into a hard cicatricial mass. At the same time the circumscribed form of gangrene may set up in the bronchi, through which the purulent matter passes, an intense form of diphtheritic or gangrenous *bronchitis*, with brown or livid discoloration, and separation of the mucous membrane, as well as relaxation and dilatation of the bronchial tubes. When the purulent matter overflows, and thus reaches a healthy part of the lung, it produces centres of bronchopneumonia, or multiple pea- or bean-sized spots of gangrene. This occurs, for example, when the gangrene commences in the upper lobe; then the purulent matter flows down into the bronchi of the lower lobe, and there gives rise to numerous fresh centres of the disease, which attain a greater or less magnitude. The *transition of circumscribed gangrene to the diffused form* is of no uncommon occurrence.

When the process has spread rapidly, severe or even fatal hemorrhage may occur from erosion of the vessels which traverse the cavity, as there is not time for the formation of thrombi, or for the obliteration of the vessels to be completed. Emboli may arise from purulent thrombi formed in the veins of the gangrenous centre, which may lead to metastatic abscesses in distant organs, such as the liver, spleen, or kidneys, etc. When the gangrenous centre is situated near the periphery of the organ, or if it has spread from the deeper parts to the surface, the pleura becomes engaged in the sloughing process, it softens, becomes perforated, and the purulent matter escapes into the cavity of the thorax. In this way a purulent and often fatal pleuritis is set up, or, if the cavity communicates with a large bronchus, pyopneumothorax is established. This result may, of course, be prevented by previous pleural adhesions, and if the gangrene then attacks the pleura, the process may extend to the wall of the thorax and the diaphragm, and open either into the cavity of the abdomen, or upon the external surface of the body. In the latter case subcutaneous emphysema may be produced (Weinberger), or the purulent matter may, as Stokes has described, follow the course of the connective tissue as far down

as the scrotum. If the opening penetrate the skin, and the contents of the gangrenous cavity be then sufficiently evacuated, complete recovery may follow, as has been observed by Rokitansky.

Diffuse gangrene, which occurs less frequently, may either proceed from the circumscribed form, or, from the very start, may be developed in a diffuse manner. In the latter case it is usually formed from gangrenous inflammation of the lung tissue in the neighborhood of a cavity containing a putrid mass, the cavity itself being either a dilated bronchus or a space in the lung tissue. More rarely it proceeds from pneumonia with purulent infiltration. In this case the parenchyma presents a tissue infiltrated with discolored and stinking fluid, and broken down into brownish-green or blackish, boggy, shreddy, or pap-like masses, which have no accurate line of demarkation, but gradually merge, through the medium of a zone of purulent infiltration, into hepatized, œdematous, or healthy lung tissue. Within such a centre of mortification one often finds one or more cavities filled with stinking, discolored ichor.

Diffuse gangrene appears to occur more frequently in the upper than in the lower lobe, and attacks the greater part of the lobe, sometimes even the whole of it. Banks reports the case of a phthisical man, æt. thirty-six, in whom the lung in its entirety was found in a state of putrefaction.

Recovery never occurs in these cases, as all attempts at reactive inflammation, which might lead to the formation of a capsule surrounding the centre of disease, are wanting. Hemorrhages, perforation of the pleura, etc., also occur here; but the patient usually dies from the severity of the general affection, without the intervention of these formidable complications. The *bronchial glands* are generally swollen, and, in case of extensive disease, transformed into masses of gangrenous softening.

Symptomatology.

Laënnec is quite justified in saying that the symptoms of gangrene of the lung are very variable, and differ essentially at different stages of the disease. This depends partly upon the

nature of the disease which generally precedes the gangrene, and partly upon its mode of spreading, the amount of tissue it destroys, and whether a communication has been established with a large bronchus. It is only by the occurrence of the last circumstance that the diagnosis is made certain, as then only are the products of the gangrenous centre brought to light through the bronchi. The sputa of these patients supply such very characteristic signs that we cannot refrain from describing them in detail.

The *sputum* which belongs to gangrene of the lung is a greenish-gray or brownish-colored fluid, with an offensive, sweetish, pungent, sickening, putrid, or even fecal smell, which sometimes disappears when the sputa have been standing some hours in the spitting-cup, as the gases which cause the smell have thus time to escape from the fluid. Forced expiration and coughing yield the same disgusting smell as the fresh sputa. Generally the smell persists throughout, but it may disappear, without the use of any special medicaments, for some days, or even weeks, and then reappear. It sometimes happens that the smell accompanying the cough precedes the expectoration of the characteristic sputa by some days.

The *amount* of the sputum thrown out is very variable ; when the secretion is abundant, it may reach from 100 to 200, or even as high as 500 Cc. According to Traube, this amount depends on the constant irritation set up in the bronchi by the passage of the ejected gangrenous masses.

Three separate layers, well defined one from the other, may be recognized in the sputum as it lies in the spitting-cup : the *uppermost*, covered with a quantity of thick froth, is of a dirty, opaque, yellowish-green color, and consists of gray or green purulent mucus here and there collected together into lumps. The *middle layer* is white, or colorless and translucent, contains a quantity of albumen, is serous in consistence, and is traversed with floating shreds of mucus. The *lowest stratum* presents a purulent sediment, green, yellowish, or often brownish in color ; in it lie soft yellow or brownish very stinking lumps, varying in size from a hemp-seed to a bean, and also many blackish flakes, probably shreds of lung tissue. Sometimes, when slight hemor-

rhages have occurred, the entire sputum is slightly colored brown, or, when copious bleeding has taken place, it may be quite black, like decomposed blood. The sputa of gangrene of the lung are then to be regarded as made up of products which come both from the gangrenous centre and from the altered bronchial mucous membrane.

Leyden and Jaffé, who instituted exact *chemical* investigations of the subject, found that the freshly ejected sputum reacts strongly alkaline. This was also the case with the distillate, and was therefore considered to depend upon volatile alkaline materials, such as ammonia and the allied organic bases. After standing a long time, an intensely acid reaction is set up by the formation of volatile fatty acids (butyric acid). Further, a number of products of the decomposition of albuminous bodies and fats can be found, such as tyrosine, leucine, margaric acid, traces of glycerine, as well as the volatile fatty acids already mentioned, and also, by the decomposition of the nitrogenous bodies, sulphide of hydrogen and ammonia. Gamgee found lactic acid, and attributes to it the fetor. The greatest share in the production of this symptom appears to belong to the butyric acid, or, according to others (Neukomm, Lebert), to valerianic or capronic acid.

Microscopic examination shows that the undermost green or yellow stratum of thick purulent matter consists of pus-cells and their detritus, and of crystals of triple phosphate. The dirty gray or blackish shreds of lung tissue are also chiefly composed of a mass of detritus, upon which are laid granular masses of yellow, brownish, or blackish pigment, and here and there a few isolated elastic fibres. The soft yellow lumps consist of numerous fat globules of various sizes, and a number of acicular crystals of margaric acid, which were first discovered by Virchow,[1] and are now found in all decomposing animal matter. According to Traube, they occur in the sputa in two distinct forms, namely, as short, fine, generally isolated crystals, and as long thick ones, collected together into bundles, which, by the pressure of the cover glass, easily become varicose. Besides

[1] Virchow's Arch., B. I., p. 334.

these, pigment, probably formed from the coloring matter of the blood, and, more rarely, some elastic fibres may be found. From a superficial examination one would think the entire stratum made up of detritus composed of finely granular substance. In this part Fischer[1] found, in a case of Traube's, numerous partly rod-shaped, partly rounded vibriones, undergoing active movements; to these Traube attributes the production of the decomposition in the bronchi, which would appear an adoption of Pasteur's views.

The discovery of these vibriones was afterwards confirmed by Leyden and Jaffé, who, with powers of 600–1000 diameters (Hartnack), were able to demonstrate granules and rods undergoing most active movements like swarms of gnats, not only in the sputa, but also post-mortem in the bronchial tubes, which seems to disprove the idea that they are only generated in the sputum after its expectoration. According to Leyden and Jaffé, the length of the rods reaches 0.003–0.006 millim., the width barely 0.001 millim. Besides these, minute thallus-like threads are sometimes found, with as many as three, four, or more distinct joints, each consisting of a rod such as we have just described. Rows of granules, which are arranged in long, thready chains like a rosary, are also found. With the addition of iodine the granules, and, in part, the rods and the adhesive material connecting them, as well as the contents of the thallus threads, become colored, while their limiting membranes remain free.

The color is yellowish-brown or purplish-red, but seldom clear blue. The coloring, their structure, as also their peculiar swarming movements, entitle us to regard these structures as plants, namely, *fungi*. Yet, according to Jaffé, the substance which is colored is neither cellulose nor amylum, since neither treating it with concentrated sulphuric acid, nor boiling with weak sulphuric acid, nor digesting with saliva, can convert it into sugar. In like manner, the failure of the xanthoproteic acid test speaks against its being a proteinic substance. Leyden and Jaffé describe the fungus as *Leptothrix pulmonalis*, and regard it as a degenerative derivative of Leptothrix buccalis, as both their granules and their rods correspond pretty well, and the same reaction with iodine occurs with both.

Besides the above, the same authors found another delicate fungus[2] formed of a thread spirally coiled up, the elements of which manifest most active, wavy, cilia-like movements, in which they sometimes appear to draw themselves together. These give no reaction with iodine, and they are not found with Leptothrix buccalis in the mucus of the mouth, but Leyden and Jaffé found them once in the dejections of a child suffering from cholera. Another less constant form is made up of pretty thick threads, consisting of two or more long joints, 0.01–0.02 mm. in

[1] Berl. klin. Wochenschr., 1864, No. 17.

[2] L. c., p. 496, Fig. 2.

length, and 0.002 mm. in breadth (l. c. Fig. 3). These undergo eel-like movements, and their nature, whether vegetable or animal, is yet doubtful.

Traube[1] has expressed the opinion, with reference to the process of decomposition, that the latter bears a close relation to rotting or putrefaction, which readily occur under the combined influence of air, moisture, and a certain temperature, in all lifeless animal tissues, which fact is supported by the experiments of Leyden and Jaffé.

They allowed ordinary purulent mucous sputum to rot in the open air, and thereby obtained changes and decompositions such as take place in pulmonary gangrene. The sputum separated itself into the well-known three layers, the smell became fetid, and in the whitish fragments of the under layer could be demonstrated free fat globules, needles of fatty acids, and a copious supply of germs and threads. But in these the iodine reaction was wanting. This, however, appears to depend less upon the kind of fungus than upon its place of origin, for even masses from gangrenous centres, which have been kept in water for some time after their removal from the body, lose their power of producing the iodine reaction.

These conditions of putrefaction are found in the stagnation of thin fluid secretions,[2] in the bronchi or bronchiectatic cavities, as also in the dead piece of lung tissue. Here the germs of the Leptothrix seem to find a soil suitable to their development, and then give rise to further decompositions.

To the *accompanying symptoms* belongs a racking *cough*, which destroys the rest at night. It is caused by the presence of the gangrenous masses that have found their way into the larger bronchi. On this account the patient lies instinctively on the affected side, with the upper part of the body high or low, according as the centre is situated in the upper or lower lobe of the lung, because in this position the putrid matter cannot flow into the sensitive bronchi, and thus the irritation producing the cough is reduced to a minimum. Rupture of a blood-vessel, and *hæmoptysis* which threatens life, may result from the intensity of the cough, but it may also be traced to the gangrenous erosion of a vessel. When such complications as pleurisy, pneumonia,

[1] Med. Klinik, 1862, p. 41.

[2] According to *Traube*, the thin fluid nature of the secretion is necessary for the production of gangrene, and to this circumstance he ascribes the fact that the hard, dry products of caseous pneumonia seldom pass into gangrene.

pyopneumothorax, or extensive infarction occur, the patient complains of *stitch in the side* and great *dyspnœa*. By means of putrid masses being occasionally swallowed, *catarrh of the stomach*, loss of appetite, and *vomiting* may come on, and very often severe and weakening *diarrhœa*. Either at the beginning or during the course of the disease, symptoms of fever appear, with very *high temperature*, rapid pulse, etc., and this condition soon passes into one of asthenia. The lightness or severity of the general symptoms is undoubtedly to be attributed to the greater or less absorption of the decomposed matter, for the fever often easily assumes a septicæmic character.

The *physical signs* afford nothing very characteristic of gangrene of the lung; the foremost is the fact, that a given part of the lung, which previously was recognized as dull and infiltrated, after a very short time yields an amphoric and metallic respiratory tone, which is well known as belonging to the formation of a pulmonary cavity. When the gangrenous centre is situated in the lower lobe, where the secretion is not constantly emptied, so that the cavity becomes quite filled, these signs disappear for a time and may again be found when the cavity is emptied. In the cases where diffuse gangrene commences with putrefaction of the secretion contained in a dilated bronchial tube, the percussion is often tympanitic at the commencement, and afterwards, indicating progressing inflammatory infiltration, it becomes non-resonant. At the same time auscultation gives at first indefinite breathing sounds, with rhonchi, and subsequently bronchial breathing, or, with the supervention of gangrenous destruction, cavernous phenomena.

Progress of the Disease.

Cases of circumscribed gangrene of the lung occur in which the patient may feel quite well for some time, even for weeks, and going about may be quite free from fever and may eat with good appetite. Gradually he becomes pale, affected by slight physical efforts, and easily tired. His friends are struck with his delicate appearance, his brilliant, sunken, and languid eye, and his weak and trembling voice, while at the same time his

pulse is small and somewhat slow. With the increasingly fetid sputa gradually appear symptoms of intense fever. Or the disease may follow another course: very severe fever, with characteristic symptoms, to be described presently, may suddenly cut short the period of immunity from suffering. Or in other cases, where the gangrene follows a hemorrhagic infarction or a circumscribed pneumonia, it may remain a considerable time unrecognized, since the symptoms of the original disease—stitches in the side, cough, dyspnœa, as also some of the physical signs—are in no way altered by the intervention of the gangrene. Doubtless suspicion may be aroused by the sudden falling off in the patient's strength, by collapse, or asthenic fever with small, frequent pulse. But the communication of the gangrenous centre with a bronchus, and the evacuation of its contents, first betray the true nature of the disease. When death follows from the primary disease, before the escape of the gangrenous mass has taken place, the gangrene may remain quite unrecognized during life. Generally speaking the disease commences with a rigor, which is commonly repeated several times, and is followed by an irregular type of fever with remissions and exacerbations. As long as the strength remains good the pulse is strong and hard, and the general constitution not much affected. When the disease is very intense (as follows an enormous absorption of purulent matter by the blood- or lymph-vessels), or when it lasts a very long time, the fever always assumes the asthenic character: the pulse becomes frequent, small, soft, and dicrotous; the patient rapidly loses strength and flesh; the face becomes pale, livid, and sunken; the tongue dry and furred, and profuse cachectic sweats appear; the appetite completely fails; colliquative diarrhœa is added, and the collapse becoming more and more obvious, an early end may be expected. Sometimes a patient falls into a typhoid condition, which usually continues until his death. Even the last described unfavorable course may occasionally be interrupted by a semblance of improvement, such as intermission of the fever, increase of strength, etc.; this, however, is generally of short duration, and the patient returns to his former condition. In these cases a copious evacuation of the gangrenous products,

or an amelioration of their injurious effects upon the general system, and a temporary cessation in the spread of the gangrene may take place, as the result in many cases of a rational mode of treatment.

Diffuse gangrene usually runs its course with the most severe general symptoms: asthenic fever, small, weak, dicrotous pulse, rapid prostration of strength, colliquative diarrhœa, and furred tongue, to which quiet delirium may be added; finally follow stupor, coma, and death. In these cases only the history of the previous illness, and the sudden turn it takes, can lead to a diagnosis; for I have often observed that when death follows quickly, as in a couple of days, and when the patient becomes rapidly collapsed, the expectoration and the characteristic fetor are wanting. Sometimes the disease is betrayed by the stinking breath emitted during coughing or on forced respiration.

Complications and Termination.

Hemorrhage may occur in the course of gangrene of the lung as the result of the erosion of a vessel which has not been obliterated. According to the size of the vessel the sputa become colored either brownish or blackish-red, like putrefying blood. The bleeding but rarely hastens death by frequent occurrence or copious effusion, and still more rarely does the patient actually bleed to death.

When situated at the periphery of the lung the gangrene may, by perforation, set up purulent and rapidly fatal *pleurisy*, which can be easily recognized by its well-known signs: friction sounds, dulness, diminished respiratory murmur, and absence of vocal fremitus, etc., etc. Or, by the simultaneous escape of air, *pneumothorax* may be developed, which commonly causes the patient to complain of rapidly increasing and severe dyspnœa and suffocation, which quickly puts an end to life. That such cases may run a favorable course, is proved by the number that are found in the literature of the subject. It has already been mentioned that a gangrenous centre may *break through the thoracic* wall, empty its contents externally, and even then the patient get well; and also that from such a perforation exten-

sive *emphysema of the cellular* tissue may occur. By means of perforation into the thoracic wall, a soft, fluctuating, often tense, elastic, discolored tumor may arise in some distant part of the body, which, by the contained air, may yield a tympanitic tone on percussion, and may be accompanied by hectic fever. The swelling may break, and give exit to its stinking contents. Recovery under such circumstances has not, so far as I know, been observed. When it perforates into the abdominal cavity severe symptoms appear: distention of the belly, intense tenderness, considerable collapse, and soon death.

The ordinary and most frequent *termination* of the disease is death, which may occur after a comparatively short time, sometimes a few days. This happens in cases of diffuse gangrene, and rapid and copious absorption of purulent matter, and in those accompanied by dangerous complications, such as purulent peritonitis, pyopneumothorax, peritonitis, etc. Generally death does not occur until during the course of the second or third week, or in some cases not until five or six weeks have elapsed. Under such circumstances, when no complication of any kind appears to cut short life, death commonly occurs from general debility and hectic. Yet cases are known, with small centres of gangrene, in which the disease may be prolonged over months, now and then undergoing a change for better or for worse, and still terminating fatally. Here the extension of the gangrene comes to a temporary standstill, the sputa lose their putrid characters and become more muco-purulent, the patient improves in strength and remains without fever until matters change again for the worse, and all the signs of gangrene reappear.

Those cases behave similarly where *imperfect recovery*, so to speak, has taken place, that is to say, when, after the evacuation of the contents, the gangrenous cavity becomes lined by a pyogenic membrane, under which circumstances tolerably good health may be enjoyed for years. During this time the patient is troubled with the constant expectoration of purulent masses; afterward, however, new putrid decomposition of the secretion in the cavity may call forth fresh inflammation, ulceration, and gangrene of its walls and their vicinity, which finally leads to a

fatal termination. In other cases of this kind, without new gangrene arising, death may take place under manifestations of hectic.

Complete recovery is a result which only occurs in cases where the centres of gangrene are small and circumscribed. The foul odor of the sputum, its peculiar grayish-green color and fluid character disappear, instead of which it becomes yellow and pus-like. With these the various other symptoms above described cease; the fever leaves the patient; the cough becomes less troublesome; the expectoration diminishes in quantity, and finally disappears altogether. A long time, at least several months, must pass over before the recovery can be regarded as perfect, and the patient quite restored.

Prognosis.

As would appear from the foregoing paragraph, gangrene is among the *dangerous* lung affections; the prognosis, however, is not absolutely unfavorable, as was formerly supposed, since recovery is not such a very uncommon occurrence.

The prognosis cannot be determined from the first in any case, since even those which are running a favorable course may sooner or later terminate fatally, as happens, for instance, in those cases in which the so-called partial recovery, with the formation of a pyogenic membrane, takes place. In *diffuse* gangrene the prognosis is positively unfavorable. In the *circumscribed* form the following points must be taken into account, in order to determine the prognosis in each individual case:

Besides the *age*, *constitution*, and *vigor* of the patient, we should take into consideration the various *causes* of this trouble, that is to say, the various diseases in the course of which the gangrene occurs. The prognosis is unfavorable in cases arising from emboli of infected or ichorous material, in those occurring after putrid bronchitis, bronchiectasis, and, under certain circumstances, after pneumonia. In the same way, diseases dangerous in themselves, preceding or accompanying gangrene, render the prognosis unfavorable.

Thus, within the last two and a half years, I have observed in my hospital practice five cases which terminated fatally in this manner:

1. Cavity of the lung. 2. Carcinoma of the œsophagus, with secondary gangrenous pneumonia. 3. Gangrenous pneumonia in a debilitated prostitute under treatment for meningitis. 4. Gangrenous pneumonia in renal disease (Morb. Brightii). 5. Case of gangrene in a debilitated man suffering from suicidal mania, who, during moderate winter temperature, having escaped from his keeper, leaped into water flowing through the yard of the hospital. The result was pneumonia, terminating in gangrene.

The *situation* of the gangrenous centre is also of importance for the prognosis. Those cases where the disease is situated in the upper lobe are more unfavorable than when the lower lobe is attacked, which doubtless depends upon the tendency of the putrid matter to flow down from the bronchi of the upper into those of the lower lobe. In such cases one may find, as has been already mentioned, numerous scattered, more or less extensive, secondary centres of gangrene in the lower lobe.

Thus Leyden and Jaffé, in the description of their seven cases, which has been frequently referred to, report that three in which the upper lobe was attacked ran a fatal course; of the remaining four, in which the gangrene was situated in the lower lobe, two cases were completely, one incompletely, cured, and the fourth was progressing favorably after the treatment.

Further, the *size* of the gangrenous centre, as may be discovered by the physical signs, the presence or absence of *fever*, as also the light character and duration of the latter, are of importance with regard to the result, as is also particularly the character of the sputum, which in favorable cases loses its fetor, diminishes in quantity, and assumes a more pus-like aspect. Lastly, the *complications* have also their importance in this question. Pleuritis, pyopneumothorax, peritonitis, partial perforation of the wall of the thorax with gravitation of the purulent matter, vomiting, and diarrhœa, render the prognosis in the highest degree unfavorable; so also does hæmoptysis, either by reducing the strength of the patient, or by its intensity, causing death directly. I must here remark that slight bleedings may occur in cases which are running a favorable course, as I have myself twice seen, when the patient feels comparatively well, and the fetid sputa are wanting. In these cases it comes from

the vascular granulations which spring up over the inner wall of the cavity, and has no signification of importance.

Diagnosis.

Small gangrenous masses, or larger ones which do not communicate with a bronchus, or which, from some other cause, cannot discharge their contents, are out of reach of diagnosis. Sometimes indeed the gangrene betrays itself by the fetor of the breath during forced expiration or coughing, before the characteristic sputum appears. Of course the condition of the mouth and fauces must be investigated by a most searching examination, to ascertain that the fetor does not depend upon carious teeth, gangrene of the mouth, tonsils, or throat, or upon ozæna. For the characteristic smell of the sputum does not necessarily point to gangrene of the lung, but only proves that a process of decomposition or putrefaction is going on in some part of the respiratory organs, which may be the interior of closed bronchi as well as the parenchyma of the lung itself, as has been shown by Dittrich and Traube, who specially called attention to the former of these conditions. In *putrid bronchitis* and in the stagnant contents of *bronchiectatic* cavities the same decomposition is found which now and then yields stinking sputa. The soft, whitish-red plugs, above described as occurring in the undermost layer of the sputum, with acicular fat crystals and leptothrix, may occur as well in one process as in the other, so long as the requirements necessary for their production are present, namely, a lengthened delay in the evacuation of the sputa. Indeed these plugs may be wanting, according to Traube, even in gangrene of the lung, when the expectoration is easily dislodged, and when a spacious, funnel-shaped cavity, with a wide opening is present. The diagnosis then may cause some embarrassment, particularly as putrid bronchitis may sometimes commence with a rigor and stitch in the side, may be accompanied by hemorrhage, and even without the intervention of gangrene may terminate fatally. In order to distinguish these affections differentially, we must bear in mind the following points. In order to determine that the decomposition takes

place in the parenchyma of the lung, and not in the uninjured bronchi, it is necessary, by means of physical examination, to prove the existence of some pulmonary disease, such as infiltration of the lung tissue, since in putrid bronchitis, or when the secretion of bronchiectasis is decomposed, dulness on percussion, bronchial breathing, sibilant rhonchi, and in the first (bronchitis) amphoric sounds are quite wanting. When the gangrenous focus, however, has a more central position, or is of small size, so that auscultation and percussion give no abnormality, the distinction is attended with much difficulty. In such cases—as indeed in general—the occurrence in the sputa of constituents of destroyed lung tissue, such as gray or blackish shreds of parenchyma, with elastic fibres arranged in the well-known manner, is of the greatest importance. Often, indeed, the elastic fibres are quite wanting, as they also may be broken up by the gangrenous process, so that one must depend on the "irregular, elongated gray shreds, of tinder-like consistence, crossed by blackish points and lines," as described by Traube, which, under the microscope, are found to be composed of "amorphous strongly refracting substance, with great quantities of thick-set but fine molecules of black pigment." The diagnosis is made quite certain when at the seat of a dense infiltration, in a short time, while the case is under observation, the signs of a cavity become developed, and fetid expectoration at the same time appears.

From *abscess of the lung* gangrene may be distinguished by the absence of the peculiar bad smell in the fresh sputum of the former, which contains less mucus, and is more simply pus-like in nature, first assuming a disagreeable odor some time after it has been ejected. In abscess, moreover, irregular villous pieces of lung tissue of a blackish color make their appearance, which, under the microscope—in contradistinction to gangrene of the lung—consist of abundant elastic tissue, with yellow-brown and blackish amorphous and crystalline pigment.

A *purulent exudation* in the pleural cavity, perforating the lung and causing stinking sputa, must be distinguished from gangrene of the lung with perforation of the pleura and pyopneumothorax by the course of the disease, and by attention to the various changes in the physical signs.

TREATMENT.

Since comparatively good results have been obtained by Scoda, Traube, Leyden, and others in the treatment of gangrene of the lung, we should not stand idly by with folded arms. Of course the earlier modes of treatment as adopted by Laënnec—leeches, cold to the head, with sinapisms and blisters—are of no value.

The *indications* for treatment are the following:

1. *Prophylaxis* comes into question, since it reminds us to attempt to diminish the amount of the secretion in bronchorrhœa and bronchiectasis, to strive against the decomposition of the secretion, and to have a regard to its timely evacuation. This is accomplished on the one hand by the well-known balsamic remedies, and upon the other by expectorants. Decomposition must be combated by improving the surrounding atmosphere by frequent ventilation, and by cleansing the mouth daily with fresh water or disinfectant remedies.

As gangrene is not an uncommon affection in lunatics, and depends partly upon inanition, partly, as many believe, upon unfavorable hygienic relations, such as overcrowding of asylums, insufficient ventilation, and want of outdoor exercise, the removal of these pernicious circumstances lies within the province of prophylactic treatment. When the patient refuses food, artificial feeding must be undertaken with great care, in order to guard against the introduction of foreign bodies into the air-passages. Also in affections of the larynx, where the patients are often nearly choked during deglutition, artificial feeding by means of an œsophageal tube is indicated.

2. In the *treatment of the disease itself* the following requirements present themselves:

For the *improvement of the expectoration*, that is to say, to *allay its fetor*, Traube found very satisfactory results from acetate of lead, which he recommends every second hour in doses of from half a grain to a grain, and with which, when the fever has considerably subsided, from three-quarters to one and a half grains of tannic acid should be taken. Others recommend chlorinated lime, from fifteen to thirty grains, in three or

four ounces of water, in tablespoonful doses; or, in the form of pill, from four to four and a half grains, with from one and a half to three grains of opium, daily (Stokes). Further, chloride of sodium (Trousseau), from two to four scruples in three or four ounces of water; also purified wood charcoal; myrrh, in powder or pill, from four and a half to nine grains at a dose; or creosote water, in tea- or tablespoonfuls; and carbolic acid, from four to fifteen grains in three ounces of water, in tablespoonfuls (Leyden), or less advantageously in pills, from a third to three-quarters of a grain four or six times a day.

The efficacy of these remedies in the above-mentioned direction is very doubtful, in spite of the manifold recommendations, and only the two last deserve attention. Whether they have any direct action on the fetid contents of the lung is not yet ascertained; for I think that neither the action (according to Leyden) of that part of the carbolic acid which is retained in the mouth, and constantly mixes with the respired air, nor its action upon the affected part of the lung after its absorption into the blood, is great enough to be taken into consideration. Its only advantage probably depends upon the fact that the small doses reaching the stomach and intestine there prevent the evil consequences which might arise from swallowing the purulent masses as above described.

The treatment by inhalation of various substances—as first introduced by Scoda for gangrene of the lung—which come into immediate contact with the diseased part of the respiratory organs, has a very different effect, and distinctly acts as a disinfectant. Although the fungus is not destroyed by this means—as has been proved by experiment by Leyden and Jaffé, who allowed among other things turpentine, chlorine, and chloride of potassium to act directly on the slough—still the effect of these remedies in diminishing the fetid sputa cannot be denied in the face of practical experience.

Up to the present time, besides the oil of turpentine, as recommended by Scoda, chlorine water, permanganate of potash, carbolic acid, oxygen, bromine (three grains each of bromine and bromide of potassium to three ounces of distilled water), have all been used with this object. With regard to the

efficacy of bromine and oxygen, the latter of which is recommended by Leyden as having given good results, I have no experience. Chlorine water can only be used in small doses (from fifteen to eighty minims in three ounces of water), since the gas evolved is irrespirable and immediately produces cough. Its action is therefore surpassed by that of other remedies. I have treated a patient with permanganate of potassa (from one and a half to fifteen grains in three ounces of water), without any result, while shortly afterwards he was freed from his disagreeable sputa by means of turpentine inhalation. The failure in treatment, in spite of its well-known disinfecting and deodorising properties, of permanganate of potassa, is probably to be explained by its ready decomposition, which takes place in the mouth and pharynx. The best and surest results are to be obtained from oil of turpentine and carbolic acid. The latter is used of the strength of from eight to fifteen grains in three ounces of water several times a day. Leyden, who recommends it, uses a two or four per cent. solution, and with sensitive patients hides the disagreeable smell and taste with mint water. With reference to the oil of turpentine we cannot share Traube's fear, which induced him to ascribe to it a peculiar injurious effect upon the reactive inflammation in the neighborhood of the gangrenous centre. This remedy can be used according to the old method, a few teaspoonfuls of it being poured on warm water or infusion of chamomile, and the vapor simply breathed. It is best administered, however, in the form of spray, to be inhaled two or three times a day, for five or ten minutes at a time, by means of an atomizer, in doses of from eight minims to half a drachm, in three ounces of water or salt solution. If pain in the head and vertigo set in, as sometimes occur with the inhalation of turpentine, or if it excites a severe cough, or if the carbolic acid is taken into the body in such quantities that the urine turns a brownish color and poisoning is feared, the use of these remedies must be discontinued and other tried means substituted instead. Oppolzer recommends an inhalation made of infusion of young pine shoots (half an ounce to three or six of water), or infusion of chamomile, linden flowers, elder flowers, peppermint, curled mint, sage, or other leaves may be also used.

When hæmoptysis occurs the inhalations are of course to be discontinued.

The indicatio morbi also imperatively demands the evacuation of the sputa out of the cavity.

3. The *general treatment* has the duty to perform of *improving the constitution* of the patient by means of nourishing diet, bitters, tonics, iron, decoctions of bark, and in purulent absorption by the use of disinfectants, such as mineral acids, quinine in large doses repeated a few times, and by alcoholic stimulants. In cases of threatening collapse, excitants such as ether, camphor, musk, are indicated. Moreover, care must be taken to provide a good, fresh, dry air, rich in oxygen, in the sick-room. For the disinfection of the sputa and the removal of the smell, which is unbearable alike for the patient and his attendants, the spitting-cup should be purified with chloride of lime, carbolic acid, permanganate of potash, and even under the bed of the patient vessels may be placed which contain weak solutions of carbolic acid, and by means of an atomizer a solution of permanganate of potash may be sprayed round the room.

4. The *symptomatic treatment* has to combat against the existing *fever*, which is best accomplished by large doses of sulphate of quinine.

A large dose of a narcotic must be administered in the evening to prevent the *cough* which often disturbs the rest at night, while in the daytime expectorants give the best results by aiding the removal of the secretion which irritates the bronchial mucous membrane. We must call attention to another point which Leyden brings forward with much justice and force, namely, in reference to the decubitus of the patient. It has already been remarked that the patient instinctively assumes such a position that the gangrenous centre will occupy the most depending part of the lung, since by this means the flowing over of the secretions into the sound bronchi, and their consequent irritation, will be avoided. For this reason care must be taken that the patient does not go about, but keeps his bed, which is particularly important for all those cases where the gangrenous centre is situated in the upper lobe. In these cases the secretion, especially in the upright position, flows down

from the bronchi of the affected part into the sound bronchi, and there sets up new centres of inflammation, which go on to gangrene.

Severe *hæmoptysis* is to be treated by the means enumerated in the chapter on that subject. Slight hæmoptysis at the granulation stage deserves but little notice.

Catarrh of the stomach, loss of appetite, and vomiting are, for the reasons above given, best treated with carbolic acid, and later with bitters. For the catarrh of the intestines, besides creosote and carbolic acid, tannic acid and quinine may be used, and to these may be added a small dose of opium. I have exhibited with good results the following prescription: ℞ Acidi tannici, quiniæ sulphatis, āā ʒiv.; creosoti, ♏xv.; opii, gr. viiss.; glycyrrhizæ, q. s. M. In pil. no. 80 div. Two pills every two hours.

NEW-GROWTHS IN THE LUNGS.

It would be beyond the province of this work, and indeed impossible, to give in it a detailed account of the extraordinarily rich literature on the subject of the new-growths which occur in the lung. We direct those who take a special interest in the subject to the handbooks of pathological anatomy and to Virchow's lectures on tumors (Berlin, 1863–1865), as well as to Schmidt's Jahrbücher der gesammten in- und ausländischen Medicin, and to Canstatt's Jahresberichte and their continuation by Virchow and Hirsch.

As a general rule, primary tumors are rare in the lung. Little is known about their mode of origin. They occur secondarily, spreading by contiguity from the neighboring organs, and pretty frequently as metastases from distant parts of the body, by means of the intervention of the blood and lymph vessels.

Pathological Anatomy.

When the new-growths dependent upon syphilis, leprosy, glanders, and lymphatic enlargements are left out of the question, the following forms occur most commonly: *Fibroma*, *Lipoma*, *Enchondroma*, *Osteoma*, *Cysts*, *Sarcoma*, *Myxoma*, and *Carcinoma*. Many of these are of little practical value, because of their small size and otherwise minor importance; they produce no symptoms of any significance for the general organism, and are only found accidentally after death. On the other hand, there are some, such as carcinoma, which show themselves by most impressive signs, and consequently must be considered in detail.

Fibromata of the size of a bean or hazel-nut are rarely met with in the lung singly, or, as in a case described by Morgan,[1]

[1] Transact. of the Pathol. Soc., 1871, XXI.—*Virchow-Hirsch*, Jahresberichte, 1871, II., p. 130.

in great numbers varying in size from that of a pin's head to that of an olive. Still more rarely are small *lipomata* found under the pleura about the size of a lentil or a pea, as rounded, slightly flattened tumors (Rokitansky). *Enchondroma* is also only found in some few isolated cases as a primary affection of the lung. More frequently it occurs secondarily to enchondromata of the bones, as rounded, irregular, notched, sharply defined tumors of the size of a pea, walnut, or egg, which are sometimes situated under the pleura, sometimes placed deeply at the root of the lung, or lying between two lobes (Lebert).[1] In the majority of cases they are purely hyaline, sometimes partially or entirely calcified,[2] or even densely ossified. Apart from the ossification of the connective tissue in carcinoma and sarcoma, *osteoma* occurs as an independent tumor, sometimes singly, sometimes multiple. One is described by Virchow[3] about the size of a man's fist, situated in the apex of the left lung, growing from the parenchyma of the organ, and only covered by pleura. There are also sometimes found numerous irregular, round, or oblong pea-sized grains of bone tissue, either on the periphery or in the interior of the lung.[4] They sometimes assume a branched form, indicating the course of the hypertrophied interlobular connective tissue, and present a strong, wiry network. Luschka[5] observed in the inferior lobes of both lungs of a man aged fifty, who had died of morbus Brightii, a region about the size of a man's hand, which was composed of a stiff network made up of bony tissue and granules like brain sand. As this network followed neither the course of the bronchi nor that of the vessels, Luschka believed it to be the remains of a partially ossified interlobular infiltration. A similar case of branched bony formation is described by Förster,[6] from Langenbeck's pathological collection in Göttingen; he doubts, how-

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[1] Physiol. Pathol., II., p. 213.

[2] *Förster*, Virchow's Archiv, XIII., p. 106.—*Lambl*, Aus dem Franz-Josef-Kinder-Spital in Prag, 1860, p. 215.

[3] Die krankhaften Geschwülste, II., p. 102.

[4] *Förster*, loc. cit., p. 105.—*Wagner*, Arch. f. physiol. Heilk., 1859, p. 411.

[5] Virchow's Archiv, Bd. X., p. 500.

[6] Ibid., Bd. XIII., p. 105.

ever, the inflammatory nature of its origin. In the neighborhood of cavities similar formations of bone may take place in the indurated tissue, either in the form of little plates or branched canals, which may be packed together so as to form large masses (Dibarder).[1]

Cysts are very rare in the lung. They generally belong to the class called dermoid cysts. The accurate examination of the individual cases suggests with much probability that they do not commence in the lung itself, but rather take their origin in the mediastinum, and subsequently grow into the lung by a gradual process. On this account they will be more accurately considered in speaking of mediastinal tumors. At the autopsy of a man, aged twenty-nine years, Schmidt[2] found, in the lower and middle lobes of the right lung, a number of cysts, some of them as large as a hen's egg, connected with one another, and containing fluid; the thin, smooth membrane forming their walls was covered by a close network of vessels. Nearly the entire left lung was transformed into a hard fibrous mass, the size of a man's fist, in which were scattered numerous cavities about as large as a bean or grape. As to the nature and mode of formation of the cysts in this case, little is known. The remains of echinococci were not found.

Myxoma is found but rarely in the lungs; on the other hand, *sarcoma* pretty frequently, both occurring only secondarily, and the latter generally when the primary seat of the growth is in the bones. For the lung is the place of selection for the metastatic appearance of *osteo-sarcoma*, as well the periosteal as the medullary, in which latter there are also found in this situation the giant cells with many nuclei, and the well-known myeloplax, and often the soft, spindle-celled sarcoma. *Carcinoma* is perhaps the form of tumor most frequently found in the lungs, the various forms—encephaloid, melanotic, scirrhus, colloid, and epithelioma—all being met with. Generally it is secondary to carcinoma of the mediastinum, breast, liver, or bones, and not uncommonly appears after the removal of a tumor from the breast, or from the bones of the upper or lower

[1] L'Union Méd., 1867, Nr. 83.

[2] Nederland. Weekblad vor Geneesk., 1851, p. 44.

extremities. Epithelioma occurs in the lung after a similar tumor of the lip, tongue, œsophagus, trachea, uterus, or vulva. Cancer is seldom primary in the lung, and the primary occurrence of epithelioma is quite denied by some writers. Recently, however, undoubted cases of cancroid have been observed by Rindfleisch[1] and Perls;[2] in the case described by the latter author secondary nodules were formed in the pleura (with hemorrhagic pleuritis) and also in the diaphragm, ribs, intestine, sphenoid bone, and choroid.

Cancer may be situated either in the deeper parts of the lung, or immediately under the pleura. When secondary it is usually developed in both lungs; when primary, commonly in one, but sometimes in both. Thus Walshe found in twenty-nine cases of primary cancer, that in eighteen only one lung was affected, in thirteen cases the right, and five the left; and Köhler, of thirty-one cases, found only one lung affected in twenty-three, of which fifteen were the right and eight the left. According to this the relation of frequency of its occurrence on the right and left sides would be about as $2\frac{1}{2}$ or 2 : 1; and in one or both lungs it would be as 1.6 or 1.3 : 1. Cancer seems to occur more frequently in men than in women. Hasse found, that out of twenty-two cases, only five were women and seventeen men. Moreover, according to him, the ages of from twenty to thirty are especially liable.

In primary cancer of the lung the growth commonly occurs in the form of a few nodules attaining the size of a cherry, walnut, hen's egg, or apple, which are often studded with extravasations of blood; it may also appear, especially in general miliary carcinosis, in very numerous small nodules the size of a pea, or even in the form of extensive infiltration of medullary cancer, which may involve a half or two-thirds of a lobe, or even one or more lobes, and thus it may acquire an appearance resembling in a striking manner gray hepatization.[3]

Doubtless in these cases the tumor takes its origin from the interlobular connective tissue, compresses the lobules and alve-

[1] Gewebelehre, 1873, III. Aufl., p. 404, Anm.

[2] Virchow's Arch., Bd. 56, p. 437.

[3] *Bierbaum*, Preuss. Vereinszeitung, N. F., 1862, Nr. 31.—*Schnyder*, Schweiz. med. Zeitschr., II., 4 and 5.—*Skrzeczka*, Virchow's Archiv, Bd. XI., p. 179.

oli, making its way into the latter, without the epithelia being converted into the elements of the tumor. According to Rindfleisch, a kind of secondary inflammation of the neighboring parenchyma, associated with a dry cellular infiltration, is added to the above in the lobules. In general the lung parenchyma is found to be compressed by the tumor, and atrophied; or when the growth is small it may remain normal in this respect, though hyperæmic, œdematous, and generally deeply pigmented. The vessels and bronchi often remain long uninjured, but they are sometimes compressed or obliterated, or filled up by cancerous masses growing into and perforating their walls. Not uncommonly a large vessel is eroded, and then severe hemorrhage may occur. The cancerous mass, according to its variety, appears as yellowish or whitish, at times soft, hard, or gelatinous nodules, from which occasionally spring thin, white canals, filled with the juice of the cancer, which follow the course of the lymph vessels. The bronchial glands, as well as the glands of the mediastinum, are filled with cancerous material.

Sometimes the masses in the lung will be found to have softened in the centre, and to have suppurated (Lebert), presenting to view extensive abscesses, or they may be found in a gangrenous condition (Stokes), or ossified. After they soften they may perforate the pleural cavity, or may open into a bronchus. Commonly the growth spreads along the pleura, more rarely along the pericardium; it then produces new nodules, or leads to the adhesion of the two layers of the pleura, or to hemorrhagic effusions into the pleural or pericardial sacs. Occasionally the growth implicates the chest wall, and burrows into the layers of its tissue. Primary cancer of the lung may give rise to secondary deposits in distant organs. When the tumor is very extensive, the heart may be pushed to one side, or backwards, while the spleen and liver may be displaced downwards. In this case the affected side of the thorax may be bulged, and the intercostal spaces prominent.

Symptomatology.

In the cases where tumors of small size and but few in num-

ber occur in the lung, or when on account of their benignity they have no marked effect on the general constitution, they run their course without symptoms. In large tumors, especially carcinoma or sarcoma, the patient complains first of a feeling of constriction and pressure in the chest, as well as of shortness of breath. The *dyspnœa* is generally at first insignificant, and only brought on by exercise. It increases gradually, affects the patient also while at rest, and finally may grow into the most intense dyspnœa or nocturnal orthopnœa. Sometimes these patients complain at the same time of a feeling of a foreign body in the throat, of pain under the sternum during the passage of food, and at the same time of the impossibility of swallowing solids. Darting or shooting pains are commonly felt in the breast or hypochondrium, and the patients are very sensitive to forcible percussion. Soon to these symptoms a *cough* is added, which is sometimes slight, sometimes distressing, and may appear in paroxysms. Generally it is dry, or accompanied by a slight frothy, catarrhal, or tenacious mucous expectoration. Sometimes the sputum is tinged with blood, but the amount of the blood is seldom considerable. One side of the *thorax is often wider* than the other, and the intercostal spaces may be obliterated. In some few cases the affected side may be diminished in size, which has been observed in cases of hard cancer, where there is great induration and retraction of the lung tissue. The affected side is *less movable* than the other.

In tolerably extensive tumors *percussion* shows an area of dulness, with irregular boundaries, on the anterior or posterior surface of the chest, accompanied by considerable increase of resistance. At the same time the heart is pushed to one side, and also the liver on the right side and the spleen on the left are pushed downwards. *Vocal fremitus* is increased. By *auscultation* weak vesicular breathing, well-marked bronchophony and bronchial breathing are heard. In certain cases, however, there are no respiratory sounds to be heard over the region of dulness, and the vocal fremitus is wanting instead of being increased. In cases of small or widely scattered tumors, no abnormality can be detected by physical examination, or at the most one may find somewhat weakened vesicular breathing, as well as dry or

moist râles, which signs are dependent upon an accompanying catarrh.

The *respiration* is often hastened, frequently loud and cooing; the voice occasionally weak or aphonic. In one case in Traube's clinique, which Fräntzel[1] reports, there occurred at times long, at times short, pauses in the respiration, some of which lasted thirty-five seconds, whereupon followed six or seven inspirations, the first of which were superficial and the subsequent ones deep, after which again came a pause.

Occasionally *cyanosis* of the face, neck, and upper part of the thorax may be observed, with great dilatation of the superficial veins, to which *œdema* of the face and neck may be added. The hands and arms swell, the patient complains of numbness and pain in the fingers, and of pain in the arm and shoulder of one or both sides, in the neck and in the back. Sometimes the pulse is irregular, and smaller in one radial artery than in the other. Pain in the head and vomiting are not uncommon. The patient is often obliged to lie on one side for a long time. Fever, emaciation, and cachectic hue of countenance appear, together with œdema of the feet and anasarca, and finally death arrives, with gradual diminution of strength, sometimes with symptoms of insufficient interchange of gases in the lung. Lebert saw in certain cases clubbing of the fingers and bending of the nails, as is observed in phthisis.

Sometimes the disease runs its course with the symptoms of phthisis. The patient has hæmoptysis and dies with hectic and profuse night-sweats (Clark).[2] In other cases dyspnœa and orthopnœa, with a severe hacking cough, appear suddenly in a person up to that time apparently quite healthy; death then occurs from increasing weakness, or may follow hæmoptysis without being preceded by any notable symptom.

Thus Lange[3] communicates the case of a laborer, æt. 63, who having previously been healthy, and having never suffered from dyspnœa, was suddenly seized with fits of choking, with severe cough, unaccompanied by expectoration, and with rapidly increasing weakness. Five centimetres from the right margin of the sternum, over

[1] Berliner klinische Wochenschrift, 1867, No. 51.

[2] The Lancet, 1856.

[3] Memorabilien, No. 3.

the entire length of the lung, there was a region of dulness the width of one's hand, which was devoid of all respiratory murmur or vocal fremitus. Nine months afterwards death occurred suddenly. At the autopsy the right lung was found studded with masses of cancer the size of an apple.

Further, Berevidge [1] mentions a case of sudden death from hæmoptysis in a man, æt. 64 years, who up to that time had appeared healthy, and only a few days before had complained of a slight cough and a feeling of oppression in the chest. At the autopsy two cancerous masses the size of a hazel-nut were found, one of which lay upon a bronchus which was ulcerated to a considerable extent. The bronchi were filled with blood.

Occasionally the disease may run the course of pneumonia, as in the diffuse or widely spread form, when an entire lobe or half a lobe is implicated by the cancerous infection.

Thus Quain [2] reports the case of a porter, æt. 27, who had sickened six months previously with slight fever, to which was added a dry cough, that steadily increased and became associated with mucous sputa. Three months after the first indisposition dyspnœa appeared. On the left side there was undoubted dulness and diminished motion, with dilatation of the affected side and bronchial breathing at the apex. After death a cancerous tumor the size of a cocoa-nut was found between the root of the left lung and the heart, as well as solitary nodules in the lung itself.

Tumors of the lung generally pursue a chronic course. As a rule carcinoma lasts from half a year to two years, seldom longer than four years. In acute carcinosis death may occur in a few weeks or months.

Analysis of the Several Symptoms.

The *dyspnœa* may be explained by the diminution of the respiratory surface, caused by the development of the tumor, by the compression and atrophy of the lung, and by the secondary inflammatory infiltration of the lobules of the lung. The more intense forms of orthopnœa may be produced by the pressure of the carcinomatous bronchial and mediastinal glands upon the trachea or upon the heart. The noisy and cooing breathing may be attributed to direct pressure on the trachea, while the

[1] Referat in Virchow-Hirsch's Jahresbericht, 1869, II., p. 109.

[2] Brit. Med. Journ., 1857, Jan. 9.

aphonia and loss of motion of the vocal cords depend upon the pressure upon one or both inferior laryngeal nerves. The *pain* in the chest arises from the pleura becoming implicated when the nodules are near the periphery of the lung, and upon secondary pleuritis with a scanty fibrinous effusion. The pain in the fingers, hand, arm, or shoulder is caused by pressure on different parts of the bronchial plexus, by means of nodules which lie external to the lung. The *cough* and *expectoration* depend upon hyperæmia of the bronchial mucous membrane and the impediment to the flow of blood in the immediate vicinity of the new-growth. In this way a slight amount of blood in the sputa is also explained; more copious supplies of blood come from the numerous vessels of the tumor, or, when the hæmoptysis is profuse, so as to cause death, from the erosion of a large vessel.

The explanation of the *physical signs* is not attended with any great difficulties, since the enlargement of one or the other side of the thorax, and the pressure on the liver or heart result from the increase in bulk of the lung; the dulness of the percussion tone and the bronchial breathing may be explained by the thickening of the lung caused by the growth. Thus also the intensified vocal fremitus is explained by the fact that the vibrations caused in the larynx and trachea by the voice are transmitted through the large bronchial tubes and communicated to the thorax wall by the condensed lung tissue. The bronchial breathing and the transmission of the vocal sounds to the thorax wall are wanting only when the tumor by its immense size also closes the larger bronchi by pressing upon them.

Cyanosis and *œdema* of the face, neck, and upper extremity only occur when tumors, for instance carcinomatous lymphatic glands, press upon the large venous trunks, such as the vena cava superior, innominata, subclavian, and jugular, and thus prevent the course of the blood to the right auricle. The increased rapidity of the pulse, without elevation of temperature, and its irregularity, are often dependent upon pressure upon the heart or great vessels. Pressure upon one or the other subclavian artery explains the unilateral weakness of the pulse, and pressure on the œsophagus, the dysphagia.

Prognosis.

When the growth has attained a size sufficient to produce a number of the above-mentioned symptoms, the prognosis is always unfavorable, since we have no means of removing the tumor. Therefore the only question is, How long can life continue under the given circumstances? The determination of this point may be possible when the nature of the tumor, its extent, and the changes produced in important organs, as well as the constitution of the patient, are clearly held in view.

Diagnosis.

Many new-growths of the lungs cannot be diagnosticated from want of important signs, and even when the latter are present the disease is not easy to recognize with certainty. For dyspnœa, orthopnœa, lancinating pain, cough and expectoration, as well as the physical signs, exhibit nothing characteristic, and occur in a number of other affections.

On account of the early hæmoptysis and certain other symptoms which belong to *phthisis*, it may be confounded with this disease. However, the form of the area of dulness caused by the tumor, and its situation in the middle of one side of the thorax, protect one against such an error, since phthisis usually commences in the apices. In the latter disease one generally finds sibilant rhonchi, and later on the signs of a cavity, with flattening or sinking in of the thorax wall over the affected part. The confusion of subacute, diffused, carcinomatous infiltration with *pneumonia* can usually be prevented by noting the want of fever in the carcinoma, as well as the absence of crepitant and sibilant rhonchi, and the characteristic pneumonic sputa. The confusion with *pleuritis* is most easily imagined, where the latter slowly and insidiously develops, and more especially in those cases where, owing to complete compression of the larger bronchi, the respiratory sounds and the vocal fremitus are similarly absent. Here an exploratory puncture may settle the point, where a combination of the two diseases does not exist. When the tumor is situated on the right side, with dulness on the right margin of the manubrium and body of the sternum, *aneurism*

must also be thought of. But the characteristic pulsation is wanting in the former, as well as the sounds of the heart and the certain differences of pulse which occur in aneurism. The diagnosis of tumor of the lung is made more certain when the above-mentioned symptoms appear in an individual who has such tumors in other parts of the body, or has had them removed by operation.

TREATMENT.

It is hardly necessary to say that this cannot be directed exactly against the disease. What we have to do in cases of tumor of the lung restricts itself simply to the alleviation of wearing symptoms, such as the cough, dyspnœa, bleeding, etc.; this we shall speak of more fully in the next chapter.

NEW-GROWTHS IN THE MEDIASTINUM.

Boerhaave, Oper. omn. med. Venet., 1733, p. 440.—*Lieutaud*, Histor. anatomica med., 1787, Vol. II., p. 236.—*Bayle*, Diction. des scien. médic., 1812.—*Nélaton*, Bulletins de la Soc. anat., 1833, p. 115.—*Bouillaud*, Traité cliniq. des mal. du cœur, 1835.—*Lobstein*, Lehrb. der pathol. Anat., übers von Neurohr, 1835, I., p. 386. Traité d'anat. pathol., I., p. 450.—*Stokes*, Diseases of the Chest, 1837.—*Mohr*, Berl. med. Centralztg., 1839, Nr. 13, u. Preuss. Vereinsztg., 1839, Nr. 26 —*Stokes*, Dublin Journ., 1842, May.—*Clark*, Lond. Gaz., 1843, April.—*Kilgour*, Lond. and Edinb. Monthly Journ., 1844, Oct.—*Tenniswood*, ibidem, July.—*Gordon*, Med.-Chir. Transact., Vol. XIII., p. 1.—*Gintrac*, Essai sur les tum. Paris, 1845.—*Little*, The Lancet, 1847, Aug.—*Demarquay*, Bullet. de la soc., 1847.—*Pfaff*, Bayer. Corr.-Blatt, 1848, pp. 50 et seq.—*Kilgour*, Monthly Journ., 1850, June.—*Briquet*, Bullet. de la société. anat., 1851, p. 409. —*Walshe*, A Practical Treatise. London, 1851, p. 364.—*Köhler*, Krebs- und Scheinkrebs-Krankheiten. Stuttgart, 1853, p. 636.—*Büchner*, Deutsche Klinik, 1853, Nr. 28.—*Obertimpfler*, Jahresbericht über die Verwaltung des Medicinalwes. im Canton Zürich, 1854.—*Nélaton*, Elements de pathol. chir., 1854, Tom. III., p. 504.—*Gordon*, The Dubl. Hosp. Gaz., 1856, Sept., No. 15.—*Laveran*, Gaz. de Paris, 1857, 9.—*Cockle*, Transact. of the Pathol. Soc. London, 1857.—*Holmes*, ibidem, Vol. IX., p. 29.—*Rogers*, Brit. Med. Jour., 1857, Dec.—*Cockle*, Med. Times and Gaz., 1858, Sept. 4.—*Murchison*, Transact. of the Pathol. Soc. of London, Vol. X., p. 240.—*Wunderlich*, Archiv. f. physiol. Heilkunde, 1858, p. 123.—*Budd*, Med.-Chir. Transact., 1859, XLII., p. 215. *Cordes*, Virchow's Archiv, 1859, Bd. XVI., p. 290.—*Fuller*, Lancet, 1860, II., 13.—*Rochard*, L'Union, 1860, Nr. 19.—*Gull*, Guy's Hosp. Rep., 3., Ser. V., p. 307.—*Cloetta*, Virchow's Archiv, 1861, Bd. XX., p. 42.—*Begbie*, Arch. of Med., 1861, II., 7, p. 145.—*Paulsen*, Hospitals Tidende, 1862, 22 ff.—*Förster*, Handbuch der spec. pathol. Anatomie, 2. Aufl., 1863, p. 278.—*Virchow*, Die krankhaften Geschwülste. Berlin, 1864, u. 1865, Bd. II., pp. 270, 376, 733 et seq.—*Pastau*, Virchow's Archiv, 1865, Bd. 34, p. 236.—*Pless*, Dissert. inaug. Göttingen, 1867.—*Jaccoud*, Leçons de cliniq. méd. Paris, 1867, p. 136.—*Holmes*, Transact. of the Pathol. Soc. of London, Vol. IX., p. 29.—*Laberge*, Oesterr. med. Jahrb., XXIV., p. 495.—*Paulicki*, Virchow-Hirsch's Jahresb., 1867, I., p. 279.—*Erichsen*, Petersburger med. Zeitschr., XII., p. 352.—*Kaulich*, Prager Vierteljahrschr., 1868, Bd. IV., p. 88.—*Helber*, Memorabilien, 1868, Lief.

8, Dec.—*Murchison*, Brit. Med. Journ., 1868, Feb. 22.—*Bonnet*, Transact. of the Path. Soc., 1868, Vol. XVIII.—*Grützner*, Dissert. Berlin, 1869.—*Feinberg*, Berl. klin. Wochenschr., 1869, Nr. 4.—*Cobet*, Neubildungen im Mediastinum. Dissert. Marburg, 1869.—*E. Albers*, Drei Fälle von Lymphosarkom. Dissert. Breslau, 1869.—*Collenberg*, Zur Entwickelung der Dermoidkystome. Dissert. Breslau, 1869.—*Riegel*, Virchow's Archiv, 1870, Bd. 49, p. 193.—*Coutagne*, Lyon. méd., 1870, Nr. 13. Virchow-Hirsch's Jahresber., 1870, II., p. 113.—*Bennet*, Brit. Med. Journ., 1870, July.—*Jephson*, Med. Times and Gaz., 1870, Sept.—*Virchow*, Dessen Archiv, 1871, Bd. 53, p. 444.—*Daudé*, Étude pratique sur les affections du médiastin. Montpellier, 1872.—*Guéneau de Mussy*, Gaz. hebd., 1871, Août 18.—*Horstmann*, Drei Fälle von Mediastinaltumor. Dissert. Berlin, 1871.—*Pöhn*, Fall von Dermoidcyste. Dissert. Berlin, 1871.—*Murchison*, Transact. of the Path. Soc., XXII., p. 7.—*Bennet*, ibidem.—*Laub*, Hosp. Tid., 1871, p. 161. Virchow-Hirsch's Jahresbericht, 1871, II., p. 111.—*Eger*, Zur Pathol. der Mediastinaltumoren. Dissert. Breslau, 1872.—*Lebert*, Klinik der Brustkrankheiten, 1874, II., pp. 650 et seq.

Etiology.

Tumors of the mediastinum are among the uncommon diseases. They occur equally frequently as primary and secondary growths. The cause of their production is unknown. There are certainly cases in the literature of the subject when after *injury*, after a severe knock or blow on the sternum, pain developed in the part, and became more and more intense, and finally the signs indicating the formation of a tumor manifested themselves (Corvisart, Gordon). In many cases it has been traced to *catching cold;* but every one now knows how much importance is often attributed, generally without careful inquiry into the subject, to this point as the origin of all imaginable diseases. *Hereditary transmission* or *predisposition* can only be accepted for some particular forms, such as certain cystic growths. The male *sex* is more frequently attacked than the female. Thus Bennet finds, in thirty-nine cases, 20 men and 19 women. Eger, in the fifty-eight cases he collected, found 37 men and 21 women. According to Riegel's collection, the relation of men to women is as 2.4 to 1.1. With regard to age, undoubtedly the young and middle-aged, from 20 to 40, are most frequently attacked. By the compilation of Pless, out of twenty-five cases eleven occurred between the ages of 20 and 30, and the next highest

number between the ages of 30 and 40. Eger found that of fifty-five cases sixteen were between 20 and 30, thirteen between 30 and 40, nine between 40 and 50, six between 50 and 60, five at the age of 60, five below 20, and one below 10 years.

Pathological Anatomy.

The greatest number of mediastinal tumors are either *carcinomata* or *sarcomata*. Cysts and simple hyperplasia of the lymphatic glands accompanying the trachea and bronchi are more rarely found. The same is true of *lymphadenomata*, which in one case of Scoda's had its origin in leucocythæmia, and of *lipomata*, which occur through hyperplasia of the normal mediastinal fat, and are associated with large collections of fat in the pericardium of obese persons. *Fibromata* and *osteomata* are the forms most rarely seen ; the latter sometimes occurring as exostoses springing from the posterior surface of the sternum. Mediastinal *cysts*, which occur but rarely, belong to the class of *dermoid cysts*. They contain numerous epithelial forms, such as hair, teeth, sebaceous and sweat glands, besides occasionally bone, cartilage, and other tissues. They may be uni- or multilocular. Since the cases occur but rarely, and are much scattered in the literature of the subject, I shall communicate briefly some of the most important of them.

Mohr found in the left lung of a girl, æt. 28 years, who had spat up hair since her sixteenth year, a very considerable cyst, which by means of a short canal communicated with the commencement of the left bronchus. On the inside of it were found several rounded knobs, here and there pedunculated, varying in size from a nut to a hen's egg, consisting of fibrous tissue provided with sebaceous and sweat glands, and from which numerous long hairs sprang. The remaining contents consisted of fatty pap with balls of hair.

The tumor which was found by Muret in Vevey, and was examined and described by Cloetta, was similar. The patient was a girl, æt. 20, who had died with the symptoms of phthisis, and who had also during her lifetime spat up hairs in great quantities. At the autopsy a dermoid cyst, the size of an apple, was found in the lower lobe of the left lung, in the fibrous walls of which were found masses of bone and cartilage. From the inner surface sprang hairs. The contents consisted of detached hairs and disintegrated fat. The cyst had its origin in the connective tissue in the neighborhood of the lung, and had grown into the latter.

Cordes's case was that of a soldier who had been previously healthy. The disease

commenced with indigestion and the symptoms of acute catarrh of the stomach; an extensive, rapidly increasing dulness developed under the sternum and in the left side of the thorax; at the end of seven weeks death occurred. Here a large sac was found in the anterior mediastinum, which communicated by an opening with the cavity of the thickened pericardium. On the inside of the wall, which was studded with plates of bone and cartilage, were found fibrous tumors the size of a nut or hen's egg. These were covered with well-formed hairs, and upon section showed acinose glands (sebaceous glands), which were connected by their stalks to hairs. The contents as usual were composed of fatty pap.

Büchner's case is remarkable from the fact that the cyst, which was the size of a child's head, was divided into two parts by a partition made up of bone and cartilage.

A tumor described by Virchow a few years ago is also interesting, as containing a mixture of various tissues. Besides a solid tumor composed of spindle-shaped and multinuclear giant cells, there were other parts which presented the characters of a multilocular cyst. The spindle cells might on the one hand be regarded as sarcoma cells, while on the other hand they were marked by numerous transverse striæ (myoma strocellulare). The cavity of the cysts was lined partly with epidermis cells and partly with ciliated epithelium. In the intermediate tissue were pieces of hyaline cartilage covered with thick perichondrium. Some parts showed a carcinomatous structure: tough, fibrous, intermediate tissue (containing a few fine spindle cells), in which round or canal-like anastomosing spaces were found, which were filled with large, somewhat angular, nucleated cells of an epithelial character. One small spot the size of a hemp-seed consisted of a mass of coarse, elastic fibres, which resembled the structure of the fetal lung.

Such cystic tumors in the mediastinum are also described by Gordon, Collenberg, Lebert, and Pöhn.

Carcinoma occurs usually in the medullary form, more rarely as *scirrhus* or *epithelioma*. Horstmann describes a case of the latter in a soldier twenty-two years of age.

Sarcoma usually takes the form of lymphosarcoma, and consists of small round cells with relatively large nuclei; rarely do they have the structure of the pure fascicular sarcoma. Many of those formerly described as soft cancer undoubtedly belong to this category. On the other hand, Virchow gives another form of tumor, of a more alveolar type, containing masses of large cells, which, however, have not an epithelial character; this must then be regarded as a mixed form—*sarcoma carcinomatosum.*

The tumors are commonly situated in the anterior mediastinum, much more rarely in the posterior, and sometimes they

are found in both. Their size is very variable. Those in the anterior mediastinum are the larger, and they occupy the entire anterior and lateral part of the thoracic cavity. They occur either as solitary tumors of greater or less size (fibroma, osteoma), or in the form of multiple small or large tumors, which still are united into a continuous mass, or, as in the case of those growths which spring from the pericardium, they form an even diffuse infiltration.

Sarcoma and carcinoma may originate in the lymph glands of the neck, or in those which accompany the trachea and bronchi, or, according to Virchow, in a persisting thymus; or, moreover, they may start from the pericardium or subpericardial connective tissue, from the periosteum of the sternum (Kaulich), and from the fat and connective tissue of the mediastinum (Riegel). Their supposed origin from the adventitia of the larger vessels appears to me to be doubtful. Much seems to justify the idea that the dermoid cysts are of fetal origin, and are developed from a dipping in of the skin of the neck, or out of the remnants of the germinal folds. With the majority of mediastinal tumors it is difficult to find out their exact point of origin, since, on account of their size, a number of important organs must be implicated by the growth. The growth of the tumor depends upon its nature. The soft, such as lymphosarcoma and medullary carcinoma, progress rapidly; the hard, on the other hand, develop slowly and remain circumscribed. The former, whether they spring from the lymphatic glands or from other tissues, usually soon attack the previously healthy lymph glands. When the tumor is situated in the anterior mediastinum it grows backwards, passes on to the pericardium, and compresses the heart and lungs; not unfrequently secondary nodules appear in the latter. Sometimes the tumor stretches itself upwards, and appears above the clavicle. Later it passes upon the trachea and primary branches of the bronchi, presses upon them from without, attacks their walls, perforates them, and then grows into the cavity of the tube. Not unfrequently the tumor implicates the large vessels, the vena cava superior, more rarely the vena cava inferior, the innominata, jugular, and subclavian veins, also the arteries (the ascending aorta, the arch of

the aorta, innominata, carotid, subclavian, and the branches of the pulmonary artery). The veins yield more readily than the arteries, since the latter, by means of their thicker walls, present greater resistance. When the tumor is situated in the posterior mediastinum, it may attack the descending aorta and the œsophagus, and may either simply compress the latter or penetrate its walls. Helber describes a case in which the œsophagus was so surrounded by the tumor that a sound could only be passed with the greatest difficulty. The constriction was four centimetres long; above and below the œsophagus was normal, as was also the mucous membrane at the point of constriction, with the exception of two lentil-sized ulcers, which were not connected with the tumor.

The narrowing of the vessels leads to a retardation in the flow of blood, and occasionally to the formation of a thrombus; collateral vascular dilatations may also occur. In cases of impediment to the flow of blood in the superior vena cava, the azygos and hemiazygos veins may swell to the thickness of the vena cava; at the same time, the collateral circulation, between the jugular and the subclavian on the one side, and the azygos and the hemiazygos on the other, becomes established through the superior intercostal veins. On account of the blood-stasis, serous effusions are produced in the pericardial and pleural sacs, and in cases of the less common compression of the inferior cava, effusion into the abdominal cavity and œdema of the lower extremities may take place. The exudations acquire, as a result of secondary cancerous growth upon the serous membranes, a more inflammatory, fibrinous, or hemorrhagic character. Occasionally the growth also implicates certain nerves, such as the trunk of the vagus, the recurrent and branches of the brachial plexus; it then either simply displaces them, or compresses, or even quite obliterates them.

The diaphragm may be pushed down, when the size of the tumor is very great, but it is very seldom that it is penetrated; also the sternum and the ribs may be bulged forward, and now and then by means of gradual ulceration they may be destroyed and broken through. Wunderlich observed in a waiter, æt. 22, an extensive lymphatic mediastinal tumor, in which the growth

was situated over the middle of the sternum, and was so flat and hard that during life it was looked upon as the bone itself which had been raised and pushed forward. In like manner, the ensiform cartilage and the attachments of the ribs were surrounded by very similar tumors.

In malignant growths metastases are sometimes found in the lungs, liver, spleen, kidneys, mammæ, etc. The soft sarcoma and carcinoma break down in the interior; not uncommonly hemorrhage into the tissue occurs from the rupture of some of the vessels.

Symptomatology.

It is almost superfluous to state that every tumor situated in the mediastinum cannot be diagnosticated during life. For the signs they produce depend solely upon their size, the position of the pressure which they exert upon the important parts in the thoracic cavity, such as the heart, lungs, trachea, bronchi, œsophagus, the large vascular trunks and the branches of nerves. Thus small tumors may exist for a long time without any symptoms at all, and first come into notice by their increase in size.

Generally the first complaint the patient makes is that of *difficulty of breathing*, which at first only comes on with energetic physical exertion, but subsequently also occurs during rest, and by degrees assumes the more exaggerated form of dyspnœa and orthopnœa, with a most unbearable sensation of suffocation, which causes the patient to seek medical aid. With great dyspnœa the frequency of respiration is only moderately increased, from twenty-four to thirty in the minute; sometimes, however, it is increased to three or four times the normal rate, and is often associated with a piping noise which may be heard at some distance.

Lancinating or pressing *pains* are frequently felt in the chest, namely, under the sternum; in the commencement these are increased by any strong bodily exertion; however, they are generally so insignificant that the patient's business is little interfered with. Occasionally a feeling of formication occurs in the fingers or hand, and there are at times severe pains in the arm, with more or less difficulty in moving it; pains are also

experienced in the neck, shoulder, and back. In other cases the pain appears only at a later stage, or the disease may run its course without any pain. After a certain time *cough* sets in, which is very distressing and spasmodic, or is characterized by scanty mucous sputa. Now and then the sputum is tinged with blood, though in cases of intense congestion severe bleeding may occur, and in one case of Church's death is said to have been caused by bleeding. The *voice* is weak, whispering, hardly audible, or it may be rough and hoarse. The patient suffers from *vertigo*, a sensation of tension in the head, *headache*, *noises in the ears*, phosphenes, and glimmering before the eyes. Upon the least movement, or even without this, *nausea* and vomiting may occur, as well as loss of consciousness, and occasionally epileptiform convulsions. Sometimes the patient complains of pain in the throat, difficulty in swallowing, a sense of resistance to the passage of the food downwards, or even an inability to swallow solids. In such cases, by means of the œsophageal bougie it may be possible to discover an impediment compressing the œsophagus behind the manubrium of the sternum; in other cases the bougie may be introduced without any difficulty. Palpitation of the heart not unfrequently exists. At the same time the pulse is pretty frequent, and sometimes upon one side weaker than upon the other, being thready, small, and hardly perceptible to the touch.

Sometimes it is impossible for the patient to lie on one or the other side. On account of the severe dyspnœa and orthopnœa he is obliged to assume the half-sitting posture in bed. The patient has an anxious expression of countenance, the eyes are prominent, the conjunctiva reddened, the pupils sometimes unevenly dilated, the thyroid gland swelled. The face may be cyanotic, more markedly so on one side than on the other, and the skin of the neck, breast, back, and one of the upper extremities may be so discolored that the hand and fingers may be quite livid. The jugular veins and other superficial veins of the neck, breast, back, and in certain cases those of the abdomen and epigastrium, are strongly developed and partially dilated into varicose enlargements. Œdema may appear in the face and trunk, in the upper, or even in the lower extremities. The hands

become stiff and cold. Even this sign usually shows a unilateral tendency, being seldom bilateral, and then always more strongly marked upon one side than upon the other.

In the great majority of cases one side of the thorax becomes wider than the other during the course of the disease; the ribs bulge forwards, and the affected part of the sternum, namely the middle and lower part, is somewhat pushed forwards. The movements of the two sides of the thorax are unequal, as the distended side moves less than the other.

Percussion gives a dull tone with increased resistance under the sternum. The dulness extends itself, on both sides, more or less beyond this limit, even reaching the axilla, and is bounded by irregular sinuous lines. It may extend upwards, as far as to the manubrium of the sternum and to the clavicle, and downwards, generally tapering off towards the slightly increased area of præcordial dulness, from which, as well as from that due to the presence of the liver, it may easily be distinguished. In very extensive tumors, which occupy the entire anterior aspect of the thorax, the area of dulness passes over without any line of demarkation into that caused by the presence of the heart and liver. Change in position causes no change in shape of the area of dulness. The heart may be pushed to one side, most commonly downwards and towards the left side, the impulse being easily felt or seen in the line of the axilla. In other cases, on the other hand, it may be very weak or quite wanting, when pericardial effusion exists, or when the tumor is placed between the heart and the wall of the thorax.

The *vocal fremitus* may be stronger or weaker than on the healthy side. The respiratory tone in the region of the dulness is feebly vesicular, indefinite, or weakly bronchial. With simultaneous catarrh of the bronchi dry and moist râles are heard. The heart tones are as a rule weak, and a blowing sound is audible in the large vascular trunks. During the course of the disease, when the other symptoms have existed a long time, the glands above the clavicle and in the neck not uncommonly begin to swell; the glands in other parts, such as the axilla, groin, or abdomen, are rarely implicated. More rarely still does the swelling in the neck, as in Wunderlich's case, form the primary

focus of the disease, whence probably it spreads to the mediastinal lymphatic glands.

The disease generally commences insidiously, and may remain latent for a length of time. There are but few cases in the literature of the subject which had an acute beginning (Horstmann), to which class those attributed to traumatic origin may also belong. With the above-mentioned symptoms, without elevation of temperature or fever, the disease runs a chronic course stretching over several months. As a rule, one seldom meets with any improvement; on the contrary, all the existing symptoms, particularly those referable to the organs of respiration, usually increase, and the patient's strength gradually diminishes, without the production of a marked cachexia, which could be said to depend upon the specific nature of the tumor. The difficulty of breathing may reach a great height, and by means of interference with the circulation in the brain the patient finally falls into a state of stupor.

Analysis of the Several Symptoms.

The *pains* in the chest, as well as in the back, neck, and throat, arise from accompanying disease of the pleura, or from pressure upon the intercostal, internal thoracic, and mediastinal nerves as well as the anterior and posterior thoracic nerves. Pain in the arm and finger, with a feeling of numbness and formication in the latter, depends upon pressure on the brachial plexus. The *difficulty of breathing* is chiefly attributable—especially when there is great stridor—to pressure upon the trachea and large bronchi, and partly to pressure on the large vessels, and doubtless may be materially increased by the simultaneous development of bronchitis, pleurisy, pericarditis, and hydrothorax. Disease of the recurrent or vagus nerve, may also have some influence in its production. This applies to those cases in which the dyspnœa and orthopnœa appear suddenly without any obvious cause, and before the other symptoms dependent upon the tumor have attained a marked character. As a rule, there is, under these circumstances, a bilateral affection of the recurrent; for, as a result of the defi-

cient innervation and incompetency of the dilator of the glottis (the posterior cricoarytenoid muscle), the glottis remains closed during inspiration, and the entrance of air is consequently impeded. The alterations in the voice, the hoarseness and aphonia, are also explained by the interference with the function of the recurrent nerve.

Vertigo, *headache*, and *singing in the ears* are the results of impediments to the circulation in the brain, and depend in the majority of cases upon hyperæmia, caused by diminished flow of blood through the heart; in some few cases, possibly, upon early weakness of the heart's action. The *dysphagia* is caused by pressure upon the œsophagus, and does not happen very frequently, owing to the fact that the tumor is commonly situated in the anterior mediastinum. Only when it encroaches upon the posterior mediastinal space, or has its primary seat there, is there any difficulty in swallowing remarked. According to Scoda, however, the dysphagia, vomiting, and singultus may be referred to pressure upon the pneumogastric nerve, and one must have recourse to this explanation when, in spite of great impediment to the passage of food, the bougie passes down the œsophagus without difficulty, or when, post-mortem, no masses can be found pressing upon this organ.

Rossbach observed in three cases interesting changes in the pupil, which he attributed to mechanical irritation of the vagus and sympathetic. In one of these patients, whose right pupil was smaller than the left, was seen, when the dyspnœa was severe and the light moderate, at the beginning of inspiration, a pretty strong bilateral dilatation of the pupils, which increased to the end of inspiration and ceased at the beginning of expiration, then returning rapidly to the normal. Moreover, one could notably dilate the pupils and convert the iris into a narrow band by exerting tolerably firm pressure on the tumor above the clavicle, by which means the pulse invariably became smaller, weaker, and slower. In the second patient, the pupils were also dilated by pressure on the tumor; the pulse, however, became more rapid. In the third case the left pupil was as small again as the right; both reacted well to light and dilated during a deep inspiration.

Alterations of the *pulse* are not unfrequently found. Increased rapidity and irregularity depend mostly upon pressure on the vagus; the smallness of the pulse in one radial or carotid comes from pressure on the large trunks, such as aorta, innominata, carotid, or subclavian. From this also comes the hypertrophy of the left heart that is sometimes met with; for, owing to the increased resistance caused by the pressure upon the above-named vessels, its force must be increased.

Cyanosis and *œdema* of the upper half of the body are the result of pressure upon the veins. Sometimes thrombi are found in the superior vena cava, the innominata, subclavian, or azygos veins, produced by the retardation of the circulation, or by the tumor growing in through the implicated wall of the vessel. When the circulation in the superior vena cava is interfered with, the blood may reach the heart by the dilatation of the superior intercostal vein, through the azygos or hemiazygos, or by the more circuitous route of the dilated mammary, superior and inferior epigastrics, and the inferior vena cava. Under these circumstances œdema of the abdomen and lower extremities may occur; these symptoms may also depend upon pressure upon the inferior vena cava, or, at the end of the disease, upon hydræmia. To the disturbances of the circulation belong also the prominence of the eyeballs and the swelling of the thyroid gland, at least when this is not simultaneously attacked with the same growth (commonly carcinoma), or when it does not happen to have undergone extensive colloid degeneration. The effusions into the pleural and pericardial sacs are partly dependent upon the interference with the circulation, and partly upon the implication of the serous membranes. The common preponderance of the symptoms on one side is the result of the unequal development of the tumor in the two sides of the thorax. The bulging of the affected part of the thorax, and the limited expansion of the latter, are explained by the mode of growth of the tumor. In some few cases the affected side of the thorax may be smaller or sunken. Gordon attributes this to the nature of the growth, the soft medullary form causing dilatation, while the scirrhus produces contraction and shrinking of the wall of the thorax.

The difference in *vocal fremitus* depends upon the structure of the tumor, whether it be an even solid mass which readily transmits sound, or whether it be irregularly developed or contain within it cystic cavities. Compression of the trachea or large bronchi may also weaken the vocal phenomena.

Duration, Result, and Prognosis.

It is very difficult to fix the duration of the disease with accuracy, since the starting-point is hidden from us from want of definite symptoms, when we have not to deal with a case such as that described by Gordon, in which the first signs presented themselves after a blow upon the chest. Lebert assumes an average of thirteen months, but there are cases which run an unusually long course, extending over years, and there are others which have but a short duration. Gordon's case ended fatally in three months, Virchow's in two months. To these may be added the case of a soldier, æt. 22, recorded by Horstmann, who, during the year of the war (1870), sickened with shivering and stitch in the side, and in twenty days an area of dulness, the size of a two-thaler piece, showed itself on the right of the sternum, which after five weeks included the entire right side of the thorax. Jaccoud's case, however, has indisputably the shortest duration on record; this ended fatally eight days after the development of the subjective phenomena. There is no doubt that the variability of the duration of the disease depends upon the nature of the tumor, for the soft malignant carcinomata and sarcomata grow more quickly, and have a relatively shorter course than the harder forms of the same species, or than the fibrous, fatty, and cystic tumors. The latter, when of a dermoid nature, have certainly an embryonic origin. At what time, however, the germinal matter really begins to form a tumor, is, up to the present, unknown.

All the mediastinal tumors observed up to the present have ended fatally. Death commonly arises from the gradual increase of the more important symptoms connected with the circulation or the respiration: for instance, simultaneous pleural or pericardial effusion, or general œdema, sopor, and the symp-

toms of gradual poisoning with carbonic acid. Death occurs rarely from paralysis of the heart, or during a sudden asthmatic attack, and its most unusual cause is hæmoptysis.

From this it would appear that the *prognosis* is invariably unfavorable, since the many modes of treatment which have been so far employed have only led to an apparent improvement.

Diagnosis.

The diagnosis of mediastinal tumors is often very difficult, and indeed in those which are of small size it is impossible. Even in those cases where pressure is exercised upon one or other of the venous trunks, or upon the various nerves, and when the corresponding symptoms exist, but where there is no dulness in the region of the sternum, the diagnosis may remain uncertain for a long time. It depends chiefly upon the *physical signs:* upon the *dulness with irregular boundaries*, *weakness, or absence of the breathing sounds;* upon the *unilateral dilatation of the thorax*, and its *incomplete expansion;* moreover, upon the symptoms caused by pressure exercised upon the different nerves, such as *loss of sensibility*, *voice*, etc.; also upon *vascular compression*, the manifestations of which are cyanosis, œdema of the face, neck, and upper extremities, and weakness of the radial pulse; and above all upon the *swelling of the visible lymphatic glands* in various parts of the body. With tumors in the posterior mediastinum, the dulness will be in the back, about at the level of the scapula, and then the effects of pressure upon the œsophagus are to be looked for.

Although these symptoms may be striking, and they no doubt point to a mediastinal tumor when they are all present, there are a number of other diseases in which many of these symptoms also occur.

Above all, in a *mediastinal abscess* where the opening has not as yet occurred, the diagnosis may be attended with difficulty. Here also we find a feeling of constriction and oppression, with a dull, boring pain behind the sternum, palpitation, laryngeal irritation, nausea, cyanosis of the face, headache, vertigo, syncope, dulness on percussion, bulging of the intercostal

spaces, loss of respiratory sounds, etc., etc. In this case the etiology must be carefully considered. The abscess generally is referred to some injury, such as a blow or knock on the sternum; to the penetration of foreign bodies, such as balls, daggers, or lance points; to disease of the sternum or ribs, such as caries or fracture; to purulent collections, or centres of inflammation in the neighborhood (inflammation in the neck, abscess of the lung, empyema, etc.); more rarely to a constitutional cause, as, for instance, the so-called metastatic inflammation of the mediastinal connective tissue, during the course of severe diseases, or the idiopathic, so-called rheumatic inflammation in this region. As a rule mediastinal abscess is accompanied from first to last by deep-seated and gradually increasing pain. At times a considerable amount of fever is associated with regularly intermitting rigors. Then the abscess gradually prepares itself to open externally, and a fluctuating tumor appears, the nature of which can be positively ascertained, if further doubt exists, by means of an exploratory puncture. The opening may occur in various parts of the thorax—the abscess seldom bursts into the pleural sac—or the pus may sink down to the inguinal and lumbar regions. According to Daudé the dulness under the sternum, caused by mediastinal abscess, undergoes a change by alteration of the position of the patient.

As a rule, *aneurisms* of the ascending portion and arch of the aorta can be distinguished without great difficulty from mediastinal tumors, as there is generally diminution of resistance over the area of dulness in aneurisms, and the heart sounds are heard with greater distinctness, a bruit accompanying the first sound. Further, the interval of time between the heart beat and the radial pulse is prolonged, and under certain circumstances there is a want of synchronism between the right and left radial pulses. However, cases of mediastinal bruit are found in which the first aortic sound is associated with a murmur, as may happen when there is pressure exerted on the aorta, or when the tumor is very vascular. Pulsation may also occur when the tumor is raised by the impulse of the aorta from behind, or when the vessel is pushed forward by the tumor, or when, as occurred in Büchner's case, a communication between the aorta

and a cyst exists. In such cases, according to Bamberger[1] and Riegel,[2] the delay in the radial pulse is always wanting, as well as the even and slow distention of the sac, which occurs in aneurism, for in the pulsating cancer the pulsation only appears in certain parts, owing to the uneven distribution of the vessels, and a tumor raised by the aorta, or a cyst communicating with it, rises and falls *in toto*. Further, in aneurism the heart's impulse is increased on account of the cardiac hypertrophy usually associated with this condition, and the heart sounds are more plainly heard; while in tumors, on the contrary, the heart sounds are generally weak, and the impulse scarcely perceptible to the touch or sight.

Pericardial effusion may be distinguished from mediastinal tumors by the characteristic shape of the area of dulness. In the former the area of dulness (corresponding to that of the heart) is conical, with a broad base placed below; in the latter it is transverse, with very irregular outline, and, tapering off below, passes insensibly into the nearly normal præcordial dulness. Also the progress of the disease, the fact that pericarditis is commonly secondary to some other severe affection (pleuritis, morbus Brightii, rheumatism, etc.), the accompanying fever, and the want of the above-mentioned symptoms of pressure on the vessels and nerves give the necessary points of distinction. The diagnosis becomes difficult, indeed almost impossible, when a pericardial effusion is added to a mediastinal tumor.

The situation of the dulness prevents any confusion between this disease and chronic *infiltration of the apices* of the lungs. In tumors the dulness is confined to the sternum and its immediate neighborhood, while in infiltration, on the other hand, it is confined to the apices of the lungs, the supra- and infraclavicular spaces. Besides this, the other physical signs are almost completely reversed in the two conditions. In the infiltration one finds well-marked intensified fremitus, loud sibilant rhonchi, loud bronchial or cavernous breathing; in the tumor the vocal fremitus is diminished, and the respiratory sounds are weak or

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[1] Lehrbuch der Herzkrankheiten. Wien, 1857, pp. 422 and 423.

[2] L. c., p. 223.

quite wanting. When the situation of the dulness and the course of the disease are taken into account, a confusion with *pleural exudation* is not easy; one can only mistake it for an encapsulated effusion lying on the anterior thoracic wall.

Tumors in the posterior mediastinum may be mistaken for an *aneurism of the thoracic aorta* and for *pneumonic infiltration.* With regard to the aneurism, one must rely upon the above-mentioned facts. The shape of the dulness, the diminished respiratory sounds, the weak vocal fremitus, the course of the disease, the dysphagia, the symptoms of pressure on vessels and nerves, which are wanting in pneumonia, are sufficient to distinguish the two affections.

It may be of great importance for the prognosis to determine the anatomical nature of the tumor. We have, for sarcoma, a guide in its rapid growth, and the simultaneous affection of other glands. Of less value is the youth of the patient, and the want of cachexia, for carcinoma too—as far as we can put faith in the microscopic observations of former years—occurs at a comparatively early age, and the cachexia is not to be regarded as the immediate result of the tumor, but rather as depending upon the disturbance of the function of many important organs, which have been encroached upon by the tumor. Cystic tumors may give a fluctuating feel, or, when they are dermoid in their nature, the fact may be established beyond a doubt either by hairs being mixed up with the sputa, as in the case of Mohr and Cloetta, or by the nature of the masses which escape when the tumor is opened by means of an incision.

Pöhn describes a case, a review of which I can only reach. In a healthy man, who suffered from pain and loss of power in the right arm two years before, a tumor appeared in the right sternoclavicular articulation. A year later a similar tumor appeared on the left side in the same situation, and under the same circumstances. In the course of four years both tumors attained a considerable size, pulsated, were soft and fluctuating. The left tumor was opened and discharged a quantity of fatty masses mixed with hair.

TREATMENT.

The treatment can naturally only be directed against the symptoms, since the remedies which have been recommended

and applied for the cure of mediastinal tumors have given but unsatisfactory results. Either internally or externally the preparations of iodine or mercury are of no use. Surgical interference, that is to say, resection of single ribs, is only indicated when the tumor is relatively small, when it occupies a circumscribed space, and when it is unconnected with any important vital organs. With carcinoma and sarcoma this procedure cannot have a satisfactory result, as these forms of growths soon recur. In cystic tumors with well-marked fluctuation, puncture and iodine injections may be indicated. The most suitable line of treatment, when there is no direct means applicable, is to sustain the patient's strength with a good nutritious diet. At the same time we should endeavor to alleviate the distressing symptoms connected with the organs of respiration: the dyspnœa and asthmatic attacks chiefly by rest, by a suitable posture agreeable to the patient (commonly the half-sitting posture) and by the use of narcotics. Counter-irritants to the chest, such as hot sponges, mustard plasters and blisters, often give momentary relief. Tracheotomy as a means of relief in cases of intense dyspnœa is irrational, as the tumor pressing upon the trachea occupies too deep a situation. Venesection in cases of stasis in the veins is injurious on account of the tendency to develop hydræmia. Local depletion on the temples, in cases of cerebral congestion, may give some relief, but should be cautiously carried out for the reason just mentioned. The modes of treatment to be adopted in cases of dangerous hemorrhage are treated of in another chapter.

PARASITES OF THE LUNGS.

a. Animal Parasites.

Up to the present time the only animal parasites which have been found in the lungs are the *Echinococcus*, the *Cysticercus cellulosae*, and the *Strongylus longevaginatus*.

1. *Echinococcus.*

For the general description of echinococci, and their mode of occurrence in the organs of respiration, we refer the reader to the detailed account given by Heller in the third volume of this Cyclopædia, pp. 555–586, while here we content ourselves with bringing forward some of the more important points connected with the symptomatology and the diagnosis.

Symptoms.

The symptoms which echinococci produce depend chiefly upon their size; hence very small cysts situated in the centre of the lung may remain latent for a very long period, even an entire lifetime.

Generally the first symptom which appears is a distressing cough, at first dry, and later accompanied by mucous sputa which may be tinged with blood. To this is next added lancinating pain in one side of the thorax, in the back, in the hypochondrium, or in the epigastrium; or the patients may complain of a dull and persistent pain in the chest. In the meantime they suffer from shortness of breath, particularly when walking or undergoing any unusual bodily exercise; the respirations

become frequent, and paroxysms of dyspnœa, with a dread of suffocation, appear. Sometimes the patient is unable to lie on the sound side. He becomes emaciated, pale, loses strength, and assumes the aspect of a consumptive person. This appearance may be more accurately simulated when he expectorates from time to time considerable quantities of blood, and when the centre of the disease is proved by physical examination to be situated in the upper lobe of the lung. There are other cases which continue for a long time with a slight cough, scarcely noticed by the patient himself, and then, upon the occurrence of a severe attack of coughing, accompanied by hæmoptysis and dyspnœa, they suddenly demand attention. More rarely the disease appears as a severe attack of acute pleuritis in a person who had been previously healthy and strong. In this case we have generally to deal with the rupture of a small peripheral cyst into the pleura.

When the echinococcus cysts attain a large size, distention of the affected side of the thorax may follow, occasionally with widening and bulging of the intercostal spaces. On account of the echinococcus being situated most commonly in the lower lobe, the lower part of the thorax is chiefly affected, and, it would appear, more frequently the right side than the left. The diaphragm becomes pushed down, and the heart, according to the side occupied by the echinococcus, is displaced to the right or left side, as the case may be. The affected side moves less than the other during respiration.

According to the size and peripheral seat of the sac, *percussion* gives a more or less extensive area of dulness, with considerable increase of resistance. The dulness is generally behind and below, more rarely in the fossa supra- or infraspinata. More frequently still the apex of the lung (the supra- and infraclavicular fossæ) remains free, and the dull percussion tone begins upon or under the clavicle.[1] The vocal fremitus is diminished. The respiratory sounds are either completely lost at the seat of dulness, or the breathing may be slightly bronchial

[1] "Häufiger noch scheint die Lungenspitze, die Fossa supra- und infraclavicularis, frei zu bleiben, und die dumpfe Perkussion erst auf der Clavicula oder unter derselben zu beginnen."—*Original Text.*

in character, or indefinite; while above and beneath the area of dulness it is frequently strongly bronchial. The signs of catarrh are usually found in the neighborhood.

The explanation of these symptoms meets with no great difficulty. The bronchial phenomena (cough, rhonchi, and mucous expectoration) are the result of the lateral pressure exercised by the echinococcus, which causes a certain amount of disturbance of the circulation. The dyspnœa is produced by compression and atrophy of the lung, by pressure on the bronchi, or plugging of the latter by echinococci, by pressure of pericardial and pleuritic exudations, or by pneumothorax, etc., after perforation into the pleural cavities. The pain is caused by pressure on the internal thoracic nerves, and by the accompanying pleuritis. Profuse hemorrhages have their origin in the erosion of a large blood-vessel occurring in the suppuration and gangrenous destruction of the neighboring tissue.

Diagnosis.

None of the symptoms have anything in them which can be looked upon as characteristic of the disease, and therefore it is always difficult, and often impossible, to distinguish it from other diseases. It is commonly confounded with *pleural effusion*. Dulness on percussion over the lower and posterior part of the chest, increased resistance, diminished fremitus, weak respiratory sounds, indistinct, weak, or loud bronchial breathing, displacement of organs, dilatation of the thorax and impeded motion are the signs recognized as characteristic of pleural effusion. The distinction becomes all the more difficult as the curved outline of the upper margin of the dulness, which, by many, is considered characteristic of echinococci, is often wanting, and the area of dulness is often very considerable when the patient first comes under observation, so that the slow (in echinococcus) or the quick (in effusion) development of this sign is of little value. Of some importance is the proof that echinococci are present in other organs, such as the liver, in which case there will be dulness over the right side of the thorax. Last year there was a patient under my care in the hospital—

whose case has been described by one of my pupils[1]—who afforded me the opportunity of seeing how fallacious this symptom may be. Here there existed dulness up to the third rib on the right side, besides which there was a gradually increasing swelling of the soft parts below the tenth rib, which showed a tendency to perforation. An incision into this gave exit to a purulent fluid containing many echinococcus sacs, and an exploratory puncture, made to confirm the diagnosis, in the fifth intercostal space gave escape to a clear fluid rich in albumen. The diagnosis founded on these points and made during life was echinococcus of the liver, with pleural effusion of the right side, the correctness of which was proved at the autopsy. The suppuration of the sac of the echinococcus had in all probability a traumatic origin.

The febrile symptoms which occur in pleuritis are of some importance, as they are usually wanting in echinococcus, only occurring with suppuration of the sac and inflammation of the surrounding lung tissue.

The confusion with hydrothorax is not easy when one considers its well-known etiological peculiarities, its common occurrence on both sides, and the alteration in the shape or situation of the dulness with the change in position of the patient.

Attention has already been called to the resemblance of this disease to chronic pneumonic infiltration of the apices of the lungs, that is, to phthisis. In this disease, as a rule, the thorax is flattened and sunken, fremitus is increased, and generally low bronchial breathing and sibilant rhonchi are heard. In those cases, however, in which the echinococcus sac has become connected with a bronchus, and has emptied itself, without the necessary attention being paid to the sputa, the likeness to phthisis may be still greater. The lung may then become contracted by the collapse of the sac. The percussion tone becomes tympanitic; by opening and shutting the mouth the pitch of the tone is altered, and sometimes a cracked-pot sound may be elicited. The fremitus may be normal, or even increased, and the respiratory murmur either bronchial or amphoric. In the cases where the dulness begins under the clavicle, one must bear

[1] *Wartena*, Een geval van Echinococcus hepatis. Amsterdam, 1874.

in mind the possibility of a new-growth (cancer) in the lung or in the anterior mediastinum. For the latter the signs of pressure upon the large vessels, cyanosis and œdema of the upper part of the body, as well as the form and situation of the area of dulness under the sternum, may suffice to settle the question. The absence of pulsation prevents any confusion between the disease in question and aneurism of the aorta.

All the cases—and indeed they are the great majority—where the diagnosis is uncertain, it can only be positively decided when the sacs of echinococci, or pieces of them, with their well-known striations, scolices, or hooks, are found in the sputa, or come to view by means of an exploratory puncture. There are cases on record where the sacs of echinococci have been spat up, without their situation or existence having been discovered during life. A variable amount of hemorrhage may accompany the expectoration of the walls of the sac, which usually come away as clear, white, translucent membranes rolled up together.

Course of the Disease.

The case may progress in such a manner as to give absolutely no outward sign of its existence. The parasite may die, and the contents of the sac may be transformed by the deposit of chalk into a mortar-like mass which is only found accidentally at the post-mortem examination. In many cases of echinococcus in the lung a communication is formed between it and one of the larger bronchi; the sac then collapses, and recovery may take place. Sometimes, after days and weeks of spitting fragments of the parasite, and an apparent amelioration of the disease, a kind of relapse occurs. The cough, pain, and dyspnœa increase, a fresh evacuation of echinococcus membrane takes place, which is followed by improvement and possibly even by permanent recovery (Lebert).[1] Often the patient does not regain his strength in spite of the removal of the cause of disease. Death occurs with fever, loss of strength, and copious purulent expectoration, and with the signs of general marasmus. In other cases perforation outwards through the wall of the thorax,

[1] Klinik der Brustkrankheiten, II., p. 671.

or through the diaphragm into the intestinal canal, may take place, and thus the sacs may be happily evacuated at stool. Perforation into the pleura, pericardium, or abdominal cavity is unfavorable, since rapid pleuritis or pyopneumothorax (where an opening into the bronchi also occurs), pericarditis or peritonitis may follow. Suppuration and gangrene sometimes occur, and usually terminate fatally. In this case the sputa become fluid, greenish-black, mixed with shreds of tissue, and stinking, and manifest the other characters of gangrenous sputa. Sudden death has been observed to follow either suffocation or profuse hemorrhage.[1]

We shall not enter here into an account of those cases where the echinococci have made their way from their seat in the abdominal cavity, in the liver, spleen, or kidneys, into the pleura, pericardium, or lung by perforating the diaphragm. In the last case evacuation through the bronchus, and indeed permanent recovery, may take place, of which fact I have observed a most striking example while assistant in Niemeyer's clinique in Greifswald.[2] The symptoms do not materially differ from those given above.

Prognosis.

The prognosis must always be guarded. Even with the complete evacuation of the sac through the wall of the chest, or by way of the bronchi, the disease is more commonly followed by death than by recovery. With regard to the treatment we must refer the reader to what is laid down in Vol. III. of this Cyclopædia.

2. *Strongylus longevaginatus* (Diesing).

This worm, which belongs to the Nematodes, has a cylindrical body, with a conical pointed head. In the vicinity of the mouth there are six pretty large wart-shaped papillæ. The length of the female reaches 26 mm., the thickness 0.7 mm.;

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[1] *Moutard-Martin*, l'Union médic., 1856, No. 78.

[2] *Noak*, De echinococcis hepatis. Dissertatio. Gryphiswaldiæ, 1860.

[3] Systema Helmintum, T. II., p. 317.

while in the male these dimensions are respectively 15-17 mm. and 0.55 mm.

These creatures were found in great numbers by Dr. Jortsits, at Clausenburg (Transylvania), in the lung substance of a boy six years old, who had died of an unknown disease, and by Rokitansky's interposition an examination was permitted. Some of the worms were loose in the lung, while others were imbedded in its parenchyma. Since this is the only observation of the kind in man, what, if any, disturbances they may create in the organs of respiration are unknown. Leuckart[1] thinks it probable that Jortsits's patient died of a parasitic form of pneumonia.

3. *Cysticercus cellulosae.*

Also the *Cysticercus cellulosae* is but rarely found in the lung, and then only a few specimens are encountered, or perhaps only a single one, even where the muscles are richly studded with them. Peculiar disturbances are not produced by their presence.

b. Vegetable Parasites.

Pneumonomycosis (Virchow).[2]

The cases in which *vegetable parasites* have been found in the human lungs are, up to the present, but few, and then only in post-mortem examinations, for they are betrayed by no symptom during life. The species most commonly observed in this situation is the *Aspergillus* (*Pneumonomycosis aspergillina*, Virchow). It was first discovered by Bennet in the tubercular masses and cavities of a phthisical patient; then it was found by Rayer and Gairdner upon the pleura in pneumothorax, and by Remak in the expectorated bronchial clots of pneumonia. Virchow[3] saw this fungus frequently in the cavities of chronic lobular pneumonia, with necrosis and softening of the tissue, and once in the bronchi of sound lungs in a girl who died of

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[1] Menschliche Parasiten. Leipzig, 1868, Bd. II., p. 404.

[2] Virchow's Archiv, Bd. IX., p. 558.

[3] Virchow's Archiv, B. IX., p. 558.

dysentery. Friedreich,[1] as well as Dusch and A. Pagenstecher,[2] observed cases of gangrene of the lung with these fungi. It would seem doubtful whether the black masses found at Greifswald, in the gangrenous walls of a pulmonary cavity, by Baum, Litzmann, and Eichstädt,[3] and considered by Schauer of Eldena as *mucor mucedo*—masses which were composed of threads entangling rounded bodies—belong to the same class. The same holds good of the fungi which Hasse and Welker found in the interior of a cancer of the lung, and that were described by Küchenmeister as *mucor mucedo*. Virchow believes that in both cases they were forms of aspergillus. According to him this growth appears either in the form of sharply defined islands, from two to three mm. in diameter, or as a thin coating of clear green or dark blackish color, and consists of thin, colorless, occasionally glistening stalks, from which greenish threads with tufted ends spring.

Sarcinæ (*Merispomœdia ventriculi*, Robin) have been frequently found in the lungs without their being found in any other part of the body, not even in the stomach. The first case of *pneumonomycosis sarcinica* was observed by Virchow[4] in the dead body of a man, aged seventy years, who had died of diarrhœa and marasmus. Near a number of obsolete nests of tubercles, on the anterior inferior extremity of the upper lobe of the left lung, was found a round, blackish-brown spot, the size of a thaler, over which the pleura was raised like a watch-glass. When this prominence was perforated, stinking gas escaped. The inside of the resulting cavity had a reddish-brown or blackish shreddy appearance, and was covered with a pulpy, reddish-brown, stinking material. The branch of the bronchus leading to the cavity was tightly plugged with a similar material. This was found by microscopic examination to consist of shreds of lung tissue, numerous blood-corpuscles, and fat granules, but chiefly of perfectly colorless packets of sarcinæ. In a second case

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[1] Ibid., Bd. X., p. 510

[2] Ibid., Bd. XI., p. 561.

[3] *Sluyter*, De vegetabilibus organism. animal. Dissert. Inaug. Berl., 1847, p. 14.

[4] *Froriep's* N. Notizen, 1846, Mai, No. 825.

observed by Virchow [1] a similar condition was found in a man aged thirty-three, who had been treated for tubercle of the lung. Besides points of caseous degeneration, infiltrations, confluent cavities, and doughy thickening of the lung, there was found in the middle lobe of the right lung a set of apparently emphysematous sacs of a dark red color, about the size of a walnut or nectarine, which projected above the surface and emitted fetid gas when punctured. The contents of these cavities corresponded exactly to what was found in the cavity of the first case mentioned. A third case of this kind is described by Cohnheim.[2] He mentions in the same paper another form of fungus, which occurred in dense gray nodules the size of a filbert, and was made up of the most exquisite cellular structure. The fungus formed thickly felted mycelium upon the walls of the alveoli, from which the threads with numerous branches grew into their cavities. Slawjansky [3] found *oidium albicans* in a spongy pea-sized nodule in the lung of a patient who had died of pneumonia. Concerning the presence of *leptothrix pulmonalis* (Leyden and Jaffé), compare the chapter on Gangrene of the Lung in this volume.

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[1] Virchow's Archiv, Bd. X., p. 401.

[2] Ibid., Bd. XXXIII., p. 157.

[3] Sitzungsprotokolle russischer Aerzte.—Referat in Virchow-Hirsch's Jahresbericht, 1867, I., p. 307.

PULMONARY CONSUMPTION

AND

ACUTE MILIARY TUBERCULOSIS.

RUEHLE.

PULMONARY CONSUMPTION.

Hippocrates, Opera edit. Kühn. Lipsiæ, 1825, T. I.—*Galeni*, De methodome dendi, lib. V, cap. XI. Lipsiae, 1825, edit. Kühn. De locis affectis, lib. IV., caput 8, 11.—*Francisci Deleboe Sylvii*, Opera medica. Praxeos medicæ, lib. I.—*Theophili Boneti*, Sepulchretum sive Anatomia practica, lib. II., de respiratione læsa. Genevæ, 1679.—*Richard Morton*, Phthisiologie oder Abhandlung von der Schwindsucht. Aus dem Lateinischen übersetzt. Helmstedt, 1780.—*Joh. Bapt. Morgagni*, De sedibus et causis morborum per anatomen indagatis. Ebroduni in Helvetia, 1779, lib. II. De morbis thoracis.—*Matthew Baillie*, The Morbid Anatomy of some of the most Important Parts of the Human Body. London, 1793.—*Antoine Portal*, Observations sur la nature et le traitement de la phthisie pulmonaire. Paris, 1809, chez Léop. Collin.—*G. L. Bayle*, Recherches sur la phthisie pulmonaire. Paris, 1810, chez Gabon.—*R. T. H. Laënnec*, Traité de l'auscultation médiate et des maladies des poumons et du cœur. 4. édit. augmentée par Andral. Paris, 1837, II. Tome.—*G. Andral*, Clinique medical, 4. édit., T. IV. Paris, 1840.—*P. C. A. Louis*, Recherches anatomiques pathologiques et thérapeutiques sur la phthisie, 2. édit. Paris, 1843.—*Cruveilhier*, Note pour servir à l'histoire des tubercules pulmonaires. Bulletin de la société anatomique, T. I., 1826.—*James Clark*, Pulmonary Consumption, with an Investigation into the Causes, Nature, Prevention, and Treatment of Tuberculous and Scrofulous Diseases in General. Phila. 1835.—*William Stokes*, A Treatise on the Diagnosis and Treatment of Diseases of the Chest. Phil., 1844.—*H. Lebert*, Traité pratique des maladies scrofuleuses et tuberculeuses. Paris, 1849.—*Reinhardt*, Charité-Annalen, 1850, 1. Jahrgang. Ueber die Uebereinstimmung der Tuberkelablagerungen mit den Entzündungsprodukten.—*Barthez* et *Rilliet*, Traité clinique et pratique des maladies des enfants, 3. Tome. Paris, 1843.—*Briquet*, Recherches statistiques sur l'histoire de la phthisie. Revue médic., 1842.—*MacCormak*, On the Nature, Treatment, and Prevention of Pulmonary Consumption.—*Herm. Brehmer*, Die chronische Lungenschwindsucht, ihre Ursachen und ihre Heilung. Berlin, 1859, 2. Aufl.—*W. A. Freund*, Der Zusammenhang gewisser Lungenkrankheiten mit primären Rippenknorpelanomalien. Erlangen, 1859.—*Virchow*, Seit 1847 im Archiv; Würzburger Verhandlungen; specielle Pathologie, 1. Bd.; die krankhaften Geschwülste, 21. Vorlesung.—

Felix Niemeyer's klinische Vorträge über die Lungenschwindsucht. Berlin, 1867. —*Hérard* et *Cornil*, De la phthisie pulmonaire. Paris, 1867.—*Sales-Girons*, La phthisie et les autres maladies de la poitrine traitées par les fumigations du goudron et le medicinal Naphta. Paris, 1846.—*Förster*, Beobachtungen über Tuberkulose im Kindesalter. Jahresbericht der Ges. für Natur und Heilkunde in Dresden, 1863.—*Heyer*, Die perkutorische Grenzbestimmung der Lungenspitze. Archiv d. Heilkunde, 1863, S. 443.—*Gintrac*, Recherches sur les dimensions de la poitrine dans leurs rapports avec la tuberculisation pulmonaire. Journ. de Bordeaux, 2. Sér. Janv., 1863.—*Godwin Timms*, On Consumption, its True Nature and Successful Treatment. London, 1860.—*James Copland*, The Forms, Complications, Causes, Prevention, and Treatment of Consumption and Bronchitis. London, 1861.—*Edw. Smith*, Consumption, its Early and Remediable Stages. London, 1862.—*Empis*, De la granulie ou maladie granuleuse, etc. Paris, 1865.—*Villemin*, Cause et nature de la tuberculose. Gaz. hebdom., 2. Sér., 1865, and 2. Sér., 1866. Bulletin d'Academie de médicine, 1865 u. 1866.—*Horace Green*, A Practical Treatise on Pulmonary Tuberculosis, etc. New York, 1864.—*Panum*, Experimentelle Beiträge zur Lehre von der Embolie. Virchow's Archiv, XXV., 1862.—*Lebert* u. *Wyss*, Beiträge zur Experimentalpathologie, etc. Virchow's Arch., XL., 1867.—*C. E. E. Hoffmann*, Beiträge zur Lehre von der Tuberkulose. Deutsches Archiv, III. Bd., 1867.—*L. Waldenburg*, Die Tuberkulose, die Lungenschwindsucht und die Scrofulose nach historischen und experimentellen Studien bearbeitet. Berlin, 1869.—*Lebert*, Ueber den Einfluss der Stenose des Conus arteriosus des Ostium pulmonale und der Pulmonalarterie auf Entstehung der Tuberkulose. Berliner klinische Wochenschr., 1867.—*Colberg*, Beiträge zur normalen und pathologischen Anatomie der Lungen. Deutsches Archiv, Bd. II., 453.—*Valentiner*, Untersuchungen zur Pathologie und pathologischen Statistik der Krankheiten der Respirationsorgane. Berlin, 1867. (Klinische Wochenschrift, 1867.)—*Dührssen*, Ueber Ursachen und Heilung der Lungentuberkulose auf Madeira. Deutsche Klinik, 1866, 1867.—*J. A. Villemin*, Etudes sur la tuberculose, preuves rationelles expérimentales de sa specificité et de son inoculabilité. Paris, 1868, XI. u. 640 pp.—*Klebs*, Ueber die Entstehung der Tuberkulose und ihre Verbreitung im Körper. Virchow's Archiv, Bd. 44, 1868.—*Ullersperger*, Die Frage ueber die Heilbarkeit der Lungenphthise pathologisch und therapeutisch untersucht. Würzburg, 1867.—*Fonssagrives*, Thérapeutique de la phthisie pulmonaire. Paris, 1866.—*Hirt*, Die Krankheiten der Arbeiter, 1. Abth., 1. und 2. Theil., 1871 u. 1873.—*Hedinger*, Die Entwickelung der Lehre von der Lungenschwindsucht, etc. Tübingen, 1864.—*Hirsch*, Handbuch der historisch-geographischen Pathologie. Erlangen, 1862–64.—*Aufrecht*, Die chronische Bronchopneumonie (Lungenschwindsucht) und die Granulie (Tuberkulose). Magdeburg, 1873, 56. S.—*Ullersperger*, Die Contagiosität der Lungenphthise. Preisschrift. Neuwied, 1869.—*C. J. B.* et *Ch. H. Williams*, Pulmonary Consumption: its Nature, Varieties, and Treatment; with Analysis of One Thousand Cases to exemplify its Duration. London, 1871.—*Buhl*, Lungenentzündung; Tuberculose und

Schwindsucht, 12. Briefe. München, 1872.—*Lebert*, Veränderungen der Körperwärme im Laufe der Tuberkulose. Deutsches Archiv, IX.—*Biermer*, Prophylaxis und Behandlung der chronischen Lungenschwindsucht. Correspondenzblatt der schweizer Aerzte, Nr. 12, 1872.

The literature on the inoculability of tuberculosis up to 1869 is contained in Waldenburg's work, and after that time in Virchow's and Hirsch's "Jahresbericht," where will also be found résumés of the numerous articles which have appeared in German and foreign journals, especially during the last few years.

INTRODUCTION.

By *consumption* or *phthisis* (tabes, consumptio) is meant in pathology those diseases which are characterized by the gradual destruction of life with constantly increasing emaciation, and from which recovery is exceptional.

Such an emaciation, consumption, or waste may arise either from excessive excretion, or from diminished ingestion. In the first case the waste results from abnormal losses, such as suppurations, blennorrhœas, etc., although the supply may be the same as usual (*phthisis*). In the latter case the waste is to a certain extent at the expense of the body itself (*marasmus*, in its strict meaning).

The *phthisical consumptions* may have their starting-point in very different forms of disease, and in different organs. Thus the older pathologists distinguished between a great variety of phthisical consumptions: phthisis laryngea, bronchialis, pituitosa, pulmonum, hepatis, intestinalis, renalis, etc. The phthisis pulmonum has always been the most conspicuous form, and its connection with phthisis laryngea and phthisis intestinalis has always been recognized.

When *autopsical examinations* began again to be made, ulcerative destructions of the lungs were found, and likewise nodular indurations. The pulmonary consumption was sometimes ulcerative, sometimes nodular. Gradually smaller and smaller nodules (milia, granula) came to be recognized; then they were found more and more frequently, and the number of cases increased in which nodules, nodes, and their degenerated products were present; until at length the nodular, tuberculous, and granular species of pulmonary phthisis came to be regarded

as the dominant form, and finally all the destructive lesions observed in the lungs, larynx, intestines, etc., in persons dying of pulmonary, laryngeal, or intestinal consumption were said to come from these nodules, or at least from the same substance of which they were formed. At the same time it was found that these nodules were almost always present in the *lungs;* in fact the conviction gained ground that the oldest lesions always originated in these nodules. And as the *pulmonary* is the predominant form of *consumption*, and *produces* the other forms, so all phthisical consumption, all phthisis, came to be regarded as dependent upon the pulmonary consumption, and this also upon the formation of nodules, tubercules, or tubercular substance. *All pulmonary phthisis was tuberculous.*

This conclusion was supposed to be the ultimate truth, and for a time investigation rested; then came the microscope.

While hitherto it had required centuries before tubercles came to be recognized by the naked eye, it now needed but a few decades, with the aid of this rapidly improved instrument, in order to become acquainted with the elementary conditions of the tissue lesions involved. What was formerly regarded as tubercle was resolved into a neoplasm (genuine miliary tubercle), and an inflammation. The latter was subdivided into several varieties, and the details of the views in regard to the tissue changes, especially those of the lungs, in what had formerly been regarded as tuberculosis, became more and more complicated. The tubercle had now scarcely any share in the tuberculosis, in fact it might be produced in animals by any kind of inoculation; what right had it then to an existence? This is nearly the present position of opinion; but in opposition to this analytical tendency there is already beginning to be felt a synthetic necessity for simpler views. All this has happened chiefly in the domain of pathological histology; and since pathology has had but a small share in these revolutions, the pathological anatomists being completely at variance with the practical pathologists, it seems proper, nay, indispensable, that the exposition of phthisis, of which the pulmonary consumption is, from the stand-point of pathology, the dominant form, should not be left to be undertaken solely by the pathological anatomists.

HISTORICAL SKETCH.[1]

Pulmonary consumption has existed among men as far back as we have any historical information. Whether it has increased or diminished, we do not know; but at all events it is at the present time very widely prevalent. Hirsch states that probably two-sevenths of all deaths are now due to this disease.

Its symptoms were described with unmistakable clearness by the physicians of antiquity, especially by Hippocrates. He ascribes the affection to a suppuration of the lungs or pleuræ, and thinks that this suppuration may arise in various ways. It may proceed from the conversion of an inflammation, or of an extravasation of blood, or of an accumulation of mucus into a suppuration; it may occur in the form of a circumscribed collection of pus, or as a diffuse infiltration, and may run either an acute or a chronic course. Tubercles, as we understand them, were unknown to the ancients; even Galen recognized only suppurations and ulcerations of the lungs, by which portions of the organ are sloughed off, as if by a putrefactive process, and discharged in the expectoration. Rhazes also holds that the suppuration of the lungs may proceed from a hæmoptysis, peripneumonia, pleuritis, or an injury, and that patients die from it because the lungs cannot be treated like external parts by the knife or cautery.

Franciscus Deleboe Sylvius appears to have been the first to recognize, along with the suppurations and ulcerations of the lungs, the existence of nodes, from the softening of which cavities arise, and he speaks of larger and smaller tubercles. He connects these nodes with the pulmonary glands, and supposes that in consequence of a hereditary and constitutional disposition this form of consumption is produced by the swelling and enlargement of these glands, and their final conversion into suppurating tubercles.

Bonnet describes lungs which contained, besides cavities, countless small tubercles. As early as 1700 Mangetus compared

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[1] With the special assistance of *Waldenburg's* Die Tuberculose, etc., nach historischen und experimentellen Studien. Berlin, 1869.

the tubercles found in the lungs, liver, spleen, kidneys, etc., with millet seeds, and also described what was manifestly a miliary tuberculosis.

In Morton's Phthisiology different forms of pulmonary consumption are, it is true, distinguished from each other, *e.g.*, those which are produced by syphilis, inflammation of the lungs, hæmoptysis, and scrofula, but the node is regarded as everywhere the first step in the destructive process in the lungs. This view was again for a long time ignored, until Stark gave a more accurate description of tubercles, and showed how cavities were formed from them.

Reid derives the tubercles from coagulated lymph, which has been effused into the pulmonary vesicles, and expresses himself as directly opposed to the hitherto-prevailing opinion of the glandular nature of tubercles.

Matthew Baillie also describes, as the most frequent lesion in the diseased lungs, the presence of nodes, which are at first about the size of the head of a pin, but afterwards, by the coalescence of several, increase to larger nodes. The breaking down of these nodes into pus he regards as the cause of consumption. He also distinguishes them from glands. At the same time, however, he speaks of the more diffuse deposits as composed of scrofulous matter, although he thinks that they consist of the same substance as the nodes. In the lymphatic glands he speaks of this matter as "caseous." In many other organs, such as the kidneys, bladder, testicles, etc., this scrofulous matter may be present as well as the tubercles, and everywhere they both possess the common property of being converted into a soft caseous mass. Portal designates this matter as *tuberculous*, and says that this is the term in general use (ordinairement appelée tuberculeuse).

Bayle, who uses the expression miliary tubercle, describes a granular as well as a tuberculous phthisis. He speaks of the former variety as not at all infrequent, although it is not mentioned by writers. The granulations, he says, are entirely distinct from the miliary tubercles. He enumerates six forms of phthisis (tuberculeuse, granuleuse, avec mélanose, ulcéreuse, calculeuse, cancéreuse), and holds that the great majority of

cases belong to the first two. Out of the 900 cases reported by him 624 were tuberculous, 183 granular, 72 melanotic, and only 14 ulcerative, 4 calculous, and 3 cancerous.

Bayle declares that tubercular phthisis is a distinctly specific disease, which may, it is true, be complicated with inflammations, catarrhs, and hæmoptyses, but does not originate in them. Moreover, a chronically inflamed lymphatic gland does not become tuberculous.

Laënnec begins his anatomical description of phthisis with the statement that the tuberculous matter is developed under two principal forms, that of isolated bodies and that of infiltration. Both of these forms present several varieties, according to the stages of development. The isolated tubercles have four principal varieties, the miliary, the crude, the granular, and the encysted tubercle; while the infiltration has three, the irregular, the gray, and the yellow. In both forms the tuberculous matter is at first gray and hyaline, gradually becoming opaque and very dense. Afterwards it softens, becomes more and more fluid like pus, and is finally discharged through the bronchi, thus giving rise to tuberculous cavities.

Judging from the only signs of inflammation which were then attainable, Laënnec denied the inflammatory nature of the tuberculous matter, and particularly that pneumonia could pass into tuberculosis. He admitted, however, that pneumonia might, in many cases, hasten the transformation of already existing tubercles. He was, moreover, just as sceptical in regard to the causation of tuberculosis by bronchial catarrh, for the reason that he was unable to satisfy himself from anatomical evidence that the latter was ever directly converted into the former.

Although Andral[1] opposed Laënnec's views on many points, he subsequently adopted the opinion that tubercles were produced independently of any irritative or inflammatory process, but their presence excited a secondary inflammation, which ultimately expelled the tubercles.

Louis adopted Laënnec's views completely, and his monograph on phthisis has for a long time been the standard textbook on the subject.

[1] Note in *Laënnec*, Traité de l'auscultation, 4 édit., 1857, Tome II., p. 92 et seq.

These views of the French writers in regard to the specific nature of phthisis were also confirmed microscopically in 1844 by Lebert, who demonstrated small, irregularly oval, granular corpuscles, to which he gave the name of tubercle corpuscles, and which he regarded as characteristic of all tuberculous matter, including both the miliary tubercle and the tuberculous infiltration.

When Rokitansky, whose pathological anatomy made its first appearance in 1842 and exerted a very great influence in Germany, also declared that tubercles are neoplasms, and adopted them for both of Laënnec's two forms, it seemed as if the specific nature of phthisis were at last definitely proved. As late as 1861 he speaks of the miliary tubercle and the tuberculous infiltration as the two forms of tubercle. The latter, he says, "consists in the impaction of the texture of the lungs with a reddish, grayish-red, grayish, finely granular, stiff, tuberculous mass, sometimes involving a whole lobe, as a lobar tuberculous infiltration, but very often lobular, that is, affecting single lobules or small aggregations of the same." "The tuberculous infiltration differs from the tuberculous granulation in the fact that in the former the tuberculous substance is produced uniformly, and in so solid a form that the pulmonary structure over a large extent becomes unrecognizable and impermeable."

In the meantime Reinhardt had in 1847 shown that tubercle corpuscles may originate from pus-cells, and had thereby deprived them of their importance. By 1850 he had established the fact that many substances hitherto regarded as tubercle were identical with the products of inflammation, and Virchow had taught us to regard the caseous metamorphosis, the tubercularization, as a general process of necrobiosis in tissues and exudations. The term tubercle he limited solely to the miliary tubercle, which he still regarded as a neoplasm. What had hitherto been called tuberculous matter was now called caseous matter, and tuberculization was called caseation. The true tubercle, that is, the earlier miliary tubercle, was regarded as a neoplasm, rich in cells, of a very unstable structure, and readily disposed to caseation, in consequence of which change the tubercle becomes opaque, white, and finally yellow. The tuberculous

infiltration was defined as an inflammation, which has become modified in various directions. The caseous pneumonia, so it was held, is often a scrofulous pneumonia,[1] whose products accumulate in the bronchioles and alveoli, and there become caseous, so that the latter appear to be completely filled with the caseous mass. These are the forms of phthisical lungs described and delineated by Carswell. All the recent numerous investigations have been based upon this histological distinction between the miliary tubercle and the infiltration; but it is not too much to say that as yet they have failed to establish conclusions which have met with general acceptance.

Bayle's miliary tubercle plays, however, only a subordinate rôle in pulmonary consumption; it is an accidental secondary product. When it forms the only anatomical lesion, we have to deal with an acute infectious disease, the acute miliary tuberculosis, which does not belong to phthisis. There is probably no chronic miliary tuberculosis in the old sense of the term. Phthisis is also anatomically a chronic inflammatory disease, with intercurrent simple forms of inflammation which heal by cicatrization. But the pernicious form of phthisis is a specific variety of inflammation with the characteristic caseous metamorphosis; this inflammation is localized in different parts of the tissues, is characterized by the fact that it begins with and also produces the true histological miliary tubercle of the smallest kind, and in itself undergoes no other metamorphosis except necrosis. Its limitation and local healing is effected by a simple inflammation in the surrounding parts. The details on this point will be given in the anatomical part of this article.

ETIOLOGY.

Everything which appears to affect the health of mankind has been alleged to be a cause of pulmonary consumption; such as deficiency and poor quality of food, lack of pure air, light, warmth, and exercise; in fact, everything which impairs the nourishment of the body, induces poverty of the blood, or depresses the nervous system.

[1] *Virchow*, Die krankhaften Geschwülste, 2. Band, 1864–65, S. 600.

Strictly speaking, this statement does not include the whole truth; there are generally other concurrent or precedent conditions, which are necessary to produce this result. These conditions are partly of a general character, and partly local, pertaining to the individual; and they sometimes occur alone, sometimes in combination.

The general conditions are the so-called constitutional anomalies, which in many cases have manifested themselves even from childhood in the form of scrofula, or exist as an inherited or congenital taint without having disclosed themselves by symptoms.

How this inherited diathesis is produced, or what is its real nature, are questions beyond our present knowledge. But if the external form of the body, and mental qualities, can be transmitted in families through generations, why should this not be the case also with the conditions which produce a disposition to certain diseases? If in the former case we do not demand that the comparison be made only between children and their parents, but include also the grandchildren and nephews, why should we not observe the same rule in regard to the inheritability of disease; why do we narrow the question to asking whether the father or the mother is known to have had the same disease at the time of conception? Is not a disease often present before it can be recognized; may there not be a disposition sufficient to be transmitted, although it does not manifest itself as a recognizable disease until afterwards? And finally, what is the statistical evidence in regard to disease in the parents at the time of conception? If even a direct examination is often insufficient to decide whether a person is entirely free from phthisical disease, how much less reliance is to be placed upon his recollection of what happened probably many years before, especially when we are entirely ignorant of his powers of observation and memory. I suppose every observing physician has had abundant experience how little importance is to be attached to a person's assertion that he is in good health, even at the time he is questioned. For these reasons, statistics in regard to the percentage of inherited cases of phthisis are evidently valueless. Moreover, if to this we add the farther difficulty that there are

no statistics, and never will be any, which distinguish between miliary tuberculosis and pulmonary consumption proper, the impossibility of settling the question of heredity by statistical evidence becomes even more apparent. Our only resort, then, is to the concurrent testimony of all times, and this is so strong that no physician who makes an unbiased examination can reject it. Whole families have been destroyed by phthisis.

The inherited influence may, of course, manifest itself in various ways: sometimes in the form of a general deviation from ideal health, a "delicacy of constitution;" at other times in the form of a particular local affection of the thorax and its viscera. These manifestations may not be noticed at birth or in early childhood, but only in later years, especially at the time of more rapid growth; for example, the period of puberal development. And thus we may go on subdividing in various ways the inheritability of phthisis, and representing its influence as now greater and now less, but in my opinion it is a very important factor. Hirsch[1] states that, according to Hörlin, in the island of Marstrand, where only one person had died from consumption during seven years, there were five consumptives then living there, four of them sisters, whose mother had died from this disease.

Inasmuch as several conditions generally concur for the production of chronic diseases, the physician will often have to decide for himself whether and how far the question of inheritability enters into the problem. I believe that a systematic and persistent observation of judicious rules of life may overcome such an inherited tendency, but I have seen this fortunate result only in rare instances. I regard phthisis as belonging in a high degree to those diseases which are in some way dependent upon an inherited tendency; but in the subsequent consideration of the individual conditions which predispose to the disease, I do not intend to refer again to the question of inheritability in connection with each of these topics.

One of these conditions, which may also be inherited, is what may be called "sickliness." This expression is not synony-

[1] Handbuch der geogr. Pathologie, II., 56.

mous with weakness, although both of these physical states often co-exist. It means merely that the individual becomes ill from slight causes, often so slight that they cannot be defined. Usually in such persons one or several parts of the body are again and again the seat of disease, and form the locus minoris resistentiæ. If this be situated in the respiratory organs, frequent and long-continued catarrhs of the nose, larynx, and air-passages occur, and perhaps give rise ultimately to pulmonary diseases, which develop into phthisis. In intercurrent diseases, in the acute exanthemata, especially measles, and in pertussis, such persons are prone to a direct implication of the lungs.

Such a sickliness is more frequently seen in the children of feeble parents or of consanguineous marriages, or when one parent is much older than the other, or when there has been a large number of births rapidly following each other. But it may also be produced by coddling and bad education in children, and by similar habits of life in later years.

The same result may be brought about by want and distress, or by the sequelæ of severe diseases, especially such as are complicated with respiratory affections (typhus, measles, pertussis), by the puerperal state, and by over-lactation. These are all conditions which depress the power of resistance to external influences, and thereby induce a predisposition to pulmonary disease and phthisis.

Scrofula is another of these conditions, and may be either inherited or acquired later in life. It is especially an anomaly of childhood, and while it often continues beyond puberty, it rarely makes its first appearance after that period. It does not consist of a sickliness or feebleness of the body, but rather of a very marked disposition to certain forms of disease, which justify us in regarding the person suffering from them as scrofulous. These forms of disease are inflammations, especially of the mucous membranes and skin, the products of which are evidently abnormal in character, so that on being absorbed by the lymphatics and carried to the lymphatic glands, they there excite inflammation, or at least hyperplasia. Among the mucous membranes those of the respiratory organs are most exposed to injurious external influences, and are therefore

especially liable to various degrees of inflammation. This is very apt to lead to enlargement of the mediastinal glands, which are unfortunately to a considerable extent beyond the control of treatment. The scrofulous inflammations are characterized by an especially abundant proliferation of cells. In the lymphatic glands these proliferated cells are pressed together so compactly that they readily die, the inflammatory products become "caseous," and form infecting foci for the development of phthisical diseases.

Scrofula also often appears for the first time after recovery from certain diseases, such as the acute exanthemata, and especially measles. Vaccination has also been regarded as a cause, and probably correctly. It does not, however, seem to produce scrofula directly by the inoculation of a "scrofulous poison," but by inducing the manifestation of the hitherto latent scrofulous symptoms, through an abnormal course of the vaccine pustule and the active fever accompanying it, in the same way as other febrile diseases of children act.

It is evident, from the fact that the apices of the lungs are almost always the parts first diseased, that the conditions in this locality must be especially favorable for the production of those changes which lead to phthisis.

Among these favorable conditions undoubtedly belongs the conformation of the chest.

Freund[1] has shown that even in early youth changes occur in the cartilages of the first ribs. These cartilages may not only be very much shortened, but may also be covered with an external osseous layer, which begins to form on their anterior upper borders, sometimes on one side, at others on both sides. These ossifications may occur even during the early years of life, or the shortening may be congenital. Freund has shown, in detail, what influence these changes must have upon the diameter of the upper part of the chest in its further development, and upon its expansion in respiration. These are certainly very important facts, and they become all the more significant in connection with the origin of apex diseases, if Freund's statement be cor-

[1] Der Zusammenhang gewisser Lungenkrankheiten mit primären Rippenknorpelanomalien. Erlangen, 1859.

rect, that in cases of recovery from disease of the apex the hindrance to respiration has been frequently found to have been remedied by a fracture, and formation of a false joint, by which the necessary mobility of the first rib in inspiration has been restored. I shall refer to this point again under the head of the symptoms of this disease.

To mention another fact, which bears upon this point: Hutchinson, an English writer, has demonstrated by numerous observations on the respiration, that in a healthy person there is a certain minimum of the so-called vital respiratory capacity, varying according to the age, sex, size and weight of body; in other words, that such a person should be able to inhale a certain cubic mass of air on a full inspiration succeeding a complete expiration. This minimum various young individuals were unable to attain, but fell considerably (about 600 ccm.) below this mark, and such persons, although no disease could be detected by other modes of physical examination, subsequently became consumptive. The further confirmations of this observation force upon us the question, What is the cause of this diminished respiratory capacity?

If the chest were filled with 600 ccm. of solid matter, especially if the latter were concentrated in the upper parts of the lungs, percussion would doubtless show the fact. And even if, in all these cases, we suppose that the first ribs are immovable, the deficit would be too great. The only explanation left is, that the deficit is due to feebleness of the respiratory muscles, and that this is the efficient cause of the ensuing phthisis. What is known as the *paralytic thorax* seems to be traceable to this factor, loss of muscular power. The slender neck, the prominent clavicles, the broad depressed intercostal spaces, and the scapulæ projecting like wings show that the principal respiratory muscles have been atrophied. The main burden of respiration is, therefore, thrown upon the diaphragm in the very years when there should be a due co-operation on the part of the other muscles. The diminished respiration in the upper parts of the lungs, and the exaggerated respiration in the lower parts, resulting from this cause, serve to explain the very general fact that pulmonary consumption almost always begins in the apices of the

lungs. But there is probably still another cause in the peculiar position of these parts. They project from three to four cm. above the clavicles, and this projecting portion, being situated outside of the chest, is subjected to the pressure of the external air. The supraclavicular region sinks in during deep inspiration, and consequently the inspiratory expansion of the apices is less than that of other parts of the lungs. On the other hand, forcible expirations drive the air into these parts, and consequently when there is an imperfect expansibility of the upper part of the chest, everything which has been discharged from the alveoli into the bronchioles of this region, or which has otherwise made its way thither, has less prospect of escape and more of retention, decay, and decomposition. The extraordinary frequency with which lesions of some kind in the apices are found at autopsies is, at all events, proof that these parts are more subject to disease than all the other portions of the lungs.

It must not be supposed, however, that these are the only causes of the fact that the apices of the lungs are so often, and in phthisis almost always, the starting-point from which the morbid process advances. The distributions of the blood, the erect posture, and other factors have also their share.[1] At all events, this question, *why phthisis begins in the upper parts of the lungs*, should be answered as exhaustively as possible. I have already mentioned some of the conditions, which seem to supply a part of this answer, and which mainly refer only to the unfavorable position of the apices as compared with other portions of the lungs, whereby mechanical disturbances in the respiratory movements are produced.

Now that we have become acquainted with the general and some of the local conditions, which, as experience has shown, favor the development of pulmonary consumption, we shall understand more easily most of the other conditions which are also acknowledged to be exciting causes.

And, first of all, we may include under this head whatever depresses the resisting power of the body, whatever produces and maintains scrofulous inflammations, and whatever interferes

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[1] Compare *Rindfleisch*, Chron. and Acute Tuberculosis (next chap.).

with the mechanism of respiration, especially for the upper parts of the chest.

Bad air and *improper food* generally go together, and are the common causes of the high percentage of mortality from consumption among the poorer classes in large cities; but how much each of these factors contributes to the result we can decide only approximately. Operatives working in close rooms, and receiving good pay, may be regarded as a class who breathe bad air and yet are well nourished, although of course the nature of the occupation will make some difference. At all events, the mortality is greater among such persons than among badly nourished laborers who work in the open air. In the case of operatives something will depend also upon whether they live near the manufactory or at a distance from it, and whether they are obliged to take a longer or shorter walk every day; those operatives who live at a distance die less frequently from consumption than do those who reside nearer. In fact, it seems obvious *à priori* that disease of the respiratory organs is produced and maintained by bad air rather than by bad nourishment. Persons who are poorly fed, that is, if the lack of proper nourishment does not proceed from a morbid state of the digestive organs, are not specially liable to consumption. Vagrant beggars often attain old age, and are not apt to die from this disease. Nor is this disease at all common as a result of the frequently long continued and constantly increasing inanition, which occurs in stenosis of the œsophagus. It is evident, therefore, that the bad quality of the air has more influence than that of the food upon the development of pulmonary consumption.

Unfortunately, we are not in a position to show in what this bad quality of the air consists, and this, not because all air is or may be bad which is not good, but because only pure air is good, and where is pure air to be found in human habitations?

The investigations which have been made in regard to the diseases of operatives have demonstrated that, with few exceptions, a residence in air mixed with foreign particles, either gaseous or vaporous, or with dust of various kinds, leads to diseases of the respiratory organs, and aggravates already existing affections, or the disposition to them; moreover, that bronchial

catarrhs, inflammations, and consumption everywhere play an important part in the diseases of these operatives. Miners, grinders, and workers in turpentine suffer especially, and varnish dust is said to be particularly injurious. In these cases there undoubtedly is a definite contamination of the air, but what proportion of consumptives belong to this class? What is the nature of the contamination in the remaining majority of cases? The answer to this question must be very indefinite. The contrast among persons thus exposed, between those who live mostly in the open air and those who spend most of their time in close, narrow, and badly ventilated rooms, is undeniable; the latter suffer from consumption in an incomparably larger proportion. In these latter cases it is uncertain whether the efficient agent consists of exhalations from the human body in the form of expectorated matters, carbonic acid, ammonia, and fatty acids, or of moisture and deleterious substances arising from the soil or the walls of the dwellings. Whether such contaminations of the air injure the respiratory organs directly, or produce pulmonary diseases indirectly, perhaps by disturbing the activity of the skin, we have no means of determining; not to mention the fact that, besides these deleterious influences, there are generally others no less efficient, whose share it is impossible to separate from that of the bad air. It is a well-established fact that the frequency of consumption increases with the density of the population, and that, the world over, the mortality from phthisis is greatest in large cities, and that in these it is greatest in most thickly populated quarters. All reports from prisons, barracks, and manufactories agree as to the fact that the mortality is far greater among the occupants of such buildings than in the surrounding population; but how much of the result is due to the air, how much to the food, or to the lack of exercise cannot be determined. But because the relation of bad air to pulmonary consumption cannot be expressed in figures, are we therefore to suppose that there is no causal relation at all? Although insusceptible of such proof, the fact that bad air induces diseases of the respiratory organs, especially consumption, and aids their progress, is obvious and undeniable. Here the matter must rest; but it may be men-

tioned also that, according to Hirt,[1] the fumes of oil and putrid gases not only do not excite the development of consumption, but rather afford a protection against it.

This *contamination of the air* we find very strikingly exemplified in *school-rooms*, in which undeveloped youth with growing chests and lungs spend daily as many as six hours in a sitting posture, and often in what a sitting posture! When the carbonic acid in the air of such rooms increases from the allowable 0.5 per mille to eight or ten per mille,[2] there is always present a proportionate amount of other ingredients, which we can recognize by the sense of smell. This air is breathed by children, some with catarrh of the air-passages and others with well-marked and even far-advanced pulmonary consumption, not merely to their own disadvantage, but also to that of others. How many school-houses are there at the present day in which any precautions are taken to remedy this evil, and where are there any in which the air of the school-room is as pure at the end of the daily session as it was at the beginning?

The same remarks will apply to the air in many dwellings in which the ventilation is imperfect, or cannot be carried out at all. This is especially the case with the sleeping-rooms, and it is a noticeable fact that we still assign more airy rooms to our handsome furniture than to human beings, especially children.

It is probably a correct belief that the moisture of the air in closed rooms specially favors the occurrence of glandular enlargements, induces scrofula, and develops its more intense forms. This result is often to be seen in children during the first years of life, and not infrequently lays the foundation for a future pulmonary consumption.

Connected with the influences of the air in producing pulmonary consumption belong also those of the *climate*, under which are to be included also the conditions of the soil, the mode of life, the influence of race, etc. The investigations of Hirsch (l. c.) have shown that neither the geographical position nor the

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[1] Die Krankheiten der Arbeiter. Breslau, 1871 u. 1873.

[2] *Geigel*, Vol. I. of this Cyclopædia, German edition. This volume has been omitted from this edition on account of its very decidedly local character.—TRANSLATOR.

temperature of a region have anything to do with the prevalence of consumption. There are regions in all zones which are free from this disease, and, on the other hand, there is no zone in which it is not very prevalent. While it is very rare in Iceland, in the island of Marstrand, on the steppes of Kirghis, in the interior of Egypt, on the plateaus of Mexico, Costa Rica, and Peru, and in the interior of South Africa, it is, on the other hand, very common in Sweden, as well as in India and in Siberia, as well as in Australia and South America. On the other hand, it appears that moist air favors consumption, especially when the temperature is continuously high, or frequent and rapid changes are associated with the moisture; while with a dry air even sudden changes (steppes of Kirghis) and continuous heat (plateaus of Peru) do not induce the disease. Moreover, a uniform climate, whether cold or warm, so long as it is dry, rarely excites consumption, and this is especially the case when the temperature is uniformly low. As regards the influence of *currents of air*, certain winds, and on their account certain seasons of the year, are everywhere considered dangerous for consumptives, or, in fact, as productive of the disease. Nothing is known as to the *modus operandi* of these causes, except that they produce catarrhal and inflammatory respiratory diseases, which may become the starting-point of consumption, and that they are apt to induce a rapid increase when this affection already exists.

It may also be regarded as a fact that an *elevated position* protects against phthisis. A height of *at least* 1,800 or 2,000 feet seems to be requisite for this purpose. Phthisis is rare on the Hartz, Styrian (in Pinzgau), and Swiss mountains, also upon the Cordilleras, and the plateaus of Abyssinia and Persia. But whether this is due to low barometric conditions is as yet undetermined; perhaps the *purity of the air* may account for it. The explanation that it is owing to the deeper breathing cannot be accepted without further evidence. There is abundant evidence to show that *change of climate* has sometimes an unfavorable, and at other times a favorable influence upon consumption. For example, we find that Icelanders frequently contract the disease on removal to Denmark; and so also do negroes, who are brought from the interior to the coast or to Europe. On the

other hand, most physicians have witnessed cures which have resulted, at least partly, from a change of climate.

How far a *deficiency of light* is to be classed among the predisposing causes it is difficult to say ; it unquestionably induces anæmia, but it is always associated with other conditions, which are of themselves favorable to the disease.

Food of poor quality promotes the occurrence of phthisis, not only by inducing inanition from being taken in insufficient amount, but also by overtaxing the digestive organs, and exciting actual disease in them in consequence of its indigestible character. A diminution in quantity probably acts by producing anæmia, the long continuance of which is regarded as an important factor in the production of this disease. All considerable losses in weight, excessive fluid discharges, hemorrhages, suppurations, and the puerperal state are admitted to be forerunners of phthisis, independently of their possessing any direct tendency to produce diseases of the respiratory organs. The same is true of chlorosis, especially when it occurs at the period of rapid growth and development of the body, and when the digestive disorders, which are so common in this affection, are associated with it. Many other anæmias, such as the chronic metallic poisonings, and especially chronic arsenical poisoning, appear to have the same effect.

The *poorness in the quality of the food* exerts especially an indirect influence upon the production of consumption. Either the food taken serves rather as ballast than nourishment, and then, notwithstanding its considerable quantity, induces inanition and anæmia, or it injures the mucous membrane of the stomach and intestines, produces repeated or chronic catarrhs, and in this way diminishes the resisting power of the body. Whether ulcerative processes are directly excited by this means, and consumption is produced by infection from the discharges, is uncertain ; but there is no doubt that in individuals, who are predisposed to consumption, intestinal diseases may continue for a long time before we can discover anything abnormal in the lungs, and that after death from phthisis in such cases the intestine presents *tuberculous* ulcerations. Unless we regard as practically non-existent everything which cannot be demon-

strated during life, there certainly is a class of cases of consumption in which the pulmonary disease is not developed until after gastric and intestinal diseases, or what may be called intestinal phthisis, have continued for a long time.

From the fact that scrofula is very prevalent in the poorer classes, who live mostly upon amylaceous food, especially potatoes, we are in the habit of regarding these articles of diet as the cause of the affection. That good, well-prepared farinaceous food, in general, produces disease of the lymphatic glands is very doubtful; but this kind of farinaceous food is rarely to be had by those who make it their chief diet, and hence we can readily see how bad, improperly cooked potatoes, eaten daily in large quantities, may, in children, excite intestinal diseases and inflammatory irritation in the mesenteric, and perhaps also other glands, which may then under a variety of favoring conditions become caseous.

Although consumption may sometimes prevail (Corsica, St. Thomas) in regions where there is no scrofula, and on the other hand scrofula is often found where consumption is absent or rare (Hartz, Styria, Peru), yet the general rule holds good, that where scrofula is very prevalent phthisis is also common, and that this is true geographically as well as ethnographically; consequently the non-development of scrofula into phthisis, in the instances referred to, must depend upon conditions which directly prevent the development of the latter.

Usually, however, everything which favors scrofula favors also the production of phthisis, and at least in this indirect way improper food should also be included among the causes of this disease.

In view of the fact that the seeds of consumption are already implanted in many children at their birth, it becomes necessary to consider the general conditions in early life, which are favorable or unfavorable to the development of the disease. Among the most important of these questions is that in regard to milk.

In all times it has been the custom, when the mother was suspected of being consumptive, to prohibit her from nursing, chiefly on her own account, because lactation is regarded as exhausting. But I may be allowed to remark that this reason-

ing is not absolutely correct. Mothers who probably, from an exaggerated sense of duty, take, while nursing, twice as much food as usual, of which the child receives only a fourth part, retain a considerable surplus for themselves, and both mother and child thrive and get fat. Indeed I have known instances in which mothers, who because they belonged to consumptive families, or had been scrofulous and still remained very delicate and anæmic, had been forbidden to nurse, finally took the matter into their own hands, their maternal duty triumphing over science, and afterwards for the rest of their lives remained free from anæmia and delicacy of constitution, while the children to all appearance did well.

It has always been regarded as improper also, for the sake of the child, to select a wet nurse who comes from a consumptive family, or has a suspicious chest. But here also no evidence has been adduced that the milk of such persons can excite phthisis. This is probably not the case; but it should be mentioned that the milk of cows with the pearl-disease has been observed to induce tuberculosis not only in calves [1] but also in other animals, and even guinea-pigs.[2] Such milk is said to produce first intestinal catarrh, and then tuberculosis of the mesenteric glands, liver, and spleen.

But even if bad human or animal milk does not excite consumption directly, it certainly belongs more frequently than is commonly supposed to the category of improper food; it deranges the digestive organs, and produces anæmia, weakness, delicacy of constitution, and probably also scrofula.

Another predisposing cause of phthisis is a faulty *carriage of the body*. Its importance will be readily understood from the stress previously laid upon the part which the conformation of the chest and the respiratory muscles play in the production of disease in the apices of the lungs. The predisposition to disease to which these parts are subject, in consequence of their unfavorable situation, must necessarily be increased when they are still farther disabled by a defective mechanism of respiration. Free, deep breathing is indispensable, both for the regulation of the

[1] *Gerlach*, 2. Jahresbericht über die K. Thierarzneischule in Hannover, 1869.

[2] *Klebs*, Arch. für experimen. Pathologie u. Pharmakol., 1 S., 168.

blood-supply in the apices, and also for the removal of irritating substances lying in the alveoli and bronchi of these parts. This point is of especial importance in childhood and youth. The prolonged sitting posture on the badly constructed seats in schools is injurious, not only because it leads to spinal curvatures, but still more because it interferes with proper breathing and the development of the chest. Equally detrimental is the position of the body in the case of various manufacturing and industrial occupations, in which even children so often engage, and by the influence of which the growth of the chest is arrested. This explains also why affections of the apices, and subsequently pulmonary consumption, are so frequent among grinders, miners, weavers, tailors, shoemakers, watchmakers, and in all persons who have to devote close attention to their work with the chest in such a constrained position as to interfere seriously with full and regular respiration. Among these detrimental occupations I might include also the constant use of the microscope, which, for still other reasons, seems to have so frequently proved injurious to pathological anatomists.

The obstructions to free respiration during sleep should also be mentioned. As every one knows, many persons, and especially children, habitually sleep in positions which seriously interfere with the proper expansion of the chest. The harm thus done is aggravated by the bad air of the sleeping apartment, and in many cases by the presence of throat catarrh and enlarged tonsils, which narrow the entrance to the larynx.

The great sensation which has been made by Villemin's experiments, in which he succeeded in producing tubercles in different animals, and by the numerous repetitions of the same, especially in France and Germany, makes it necessary for us to refer to this subject. In these experiments the point involved is not pulmonary consumption, but the production of granulations, which most observers have regarded as identical with human tubercle. But if the objection which has been quite recently urged[1] be well founded, that these granulations are

[1] *Friedländer*, Ueber locale Tuberkulose. Sammlung klinischer Vorträge von Volkmann.

not tubercles in the histological sense, all the conclusions based on this hypothesis would be invalidated. If, however, we admit that the insertion of various substances into different parts of the bodies of rabbits and guinea-pigs did in reality produce miliary tubercles, this fact would only prove experimentally what we have already learned from anatomical observation in the human subject, viz., that miliary tuberculosis occurs, sometimes as a local, and at other times as an extensively diffused affection, in the course of various morbid processes attended with caseous products. In these experiments there was all the more reason for regarding this miliary tuberculosis as the result of absorption from caseous foci, because in many cases there was observed to be a migration from these foci into the surrounding tissues, that is to say, miliary tubercles were found to occur first of all at these points. On the other hand, in by far the majority of cases, whenever miliary tuberculosis was observed, caseous matter also existed in the same body. These experiments upon animals confirmed the probability of such a self-infection in man, and led also to the belief that the miliary tubercle was in general only a secondary product, and played only this part in pulmonary consumption. The result was that phthisis as such entirely lost its tuberculous character, and the victory over the Frenchman Laënnec was complete. But the pathologico-anatomical portion of this article will show that there is no necessity for entire acceptance of this opinion, and my views upon this point are shared by others who are skilled microscopists. As regards the various symptoms observed in the inoculated animals, there is no evidence whatever that the disease is identical with human tuberculosis. The animals sicken and emaciate, their hair becomes bristly, and in the worst event they die, and this is tuberculosis! These experiments are valuable, however, because they show the dangers of caseous foci, and that scrofula bears a direct relation to genuine tuberculosis as well as to consumption itself.

Scrofula in many persons not only produces a general disposition to inflammations, which run a special course and lead to consumption, but, like other diseases, it may also supply the infecting matter for miliary tubercles. These results from

inoculation renew the old question of the *contagiousness* of pulmonary consumption. A passage in Morgagni shows that the disease was formerly regarded as very contagious, and also explains why he has written so little upon phthisis: "præcipuam causam accipe brevitatis nostræ. Valsalva posteaquam juvenis in phthisis periculum venit, ut in ejus scriptum est vita, cadavera istiusmodi morbis absumptorum minus, opinor, quæsivit. Ego vero, ut me tibi aperiam, illa fugi de industria adolescens, et fugio vel senex, tunc ut mihi, nunc ut studiosæ, quæ me circumstat, juventuti prospiciam, cautius fortasse quam opus sit, at tutius. Itaque non multa ille, ego vix aliquod dissecui."

Jacobi reports that a dog which ate the sputa of his phthisical master died of consumption. Even at the present day it is commonly supposed that the disease may be communicated through the intimate relations existing between husband and wife, and although it is difficult to adduce satisfactory evidence upon this point, we may be permitted to agree with Laënnec and Andral, that caution and cleanliness in taking care of, and associating with those who are far advanced in consumption, are certainly desirable. Laënnec was convinced that he had contracted a tuberculous nodule through a wound from a saw while making an autopsy in a case of phthisis. He succeeded, however, in destroying it with the butter of antimony. As Laënnec was undoubtedly familiar with the appearances of the ordinary dissection pustules, the fact that he thought it worth while to mention this incident shows that he must have meant something different from them.

Pulmonary consumption frequently develops also in persons who have previously been attacked by other diseases, or who are still suffering from them. The connection here is twofold; in some cases the phthisis is produced indirectly in consequence of serious derangements in nutrition, anæmia, and marasmus, as, for instance, in diabetes mellitus, convalescence from severe diseases, and the more profound derangements of the digestive organs previously alluded to; in other cases the relation is more direct, as is the case when there has been antecedent disease of the respiratory organs.

This involves the question as to the connection between con-

sumption and the various forms of *bronchial catarrh*, *pneumonia*, *pleurisy*, and *hæmoptysis*.

Opposing views have always prevailed, and still prevail, on all these points, on account of the difficulty in obtaining evidence.

According to the opinion at present entertained, bronchial catarrhs are said to induce pulmonary consumption by extending to the parenchyma, and there exciting catarrhal pneumonia, which through caseation may lead to phthisis. In the great majority of bronchial catarrhs this result does not occur. These catarrhs extend in both directions, especially downwards, where the most abundant crackling râles are heard, and yet no symptoms of pneumonia occur. But if the râles are heard in one apex, or in both apices only, or are constantly present in only one lung, in other words, if the situation of the râles is different from what it usually is, then symptoms of consumption are apt to follow. Was the catarrh merely an ordinary catarrh abnormally situated, or was it conditioned by antecedent disease of a different character? Was the catarrh primary, or was it not rather secondary? If most of the cases of consumption begin with the signs of catarrh, if many patients trace the beginning of their disease to a cold, is this a sufficient reason for supposing that the phthisis actually started from an extension of a simple catarrhal inflammation to the parenchyma? If this is the case, why do not the capillary catarrhs, which are from the start situated nearest to the parenchyma, terminate in phthisis? It is well known that the opposite is the rule. Phthisis is not introduced by the ordinary diffuse catarrhs, but by catarrhs which have an exceptional character from the start; exceptional in their situation, which makes it probable that their localization in the apices was produced by parenchymatous disease which we are unable to detect by our present means of diagnosis; exceptional because they occur in persons in whom there is a special tendency to disease of the apices in consequence of inherited predisposition to scrofula, formation of the chest, occupation or habitual position of the body; and exceptional also, perhaps, because they are dependent upon certain diseases, such as measles and pertussis, all, or at least several, of which conditions

are not infrequently met with at the same time in the same individual.

A person may have several attacks of bronchial catarrh year after year without becoming consumptive, although the catarrh is left entirely to its own course, and many catarrhs last for years without resulting in phthisis; in fact, it seems as if the longer they continue the less is the danger of this result.

Bronchial catarrh should not be regarded as *in itself* a cause of consumption; it produces this result only under *certain conditions* which induce the disease even when there is no catarrh.

Is *pneumonia* a cause of consumption? Here, also, the same remarks will apply as in the case of catarrh. At all events croupous pneumonia very rarely leads directly to consumption, and many anatomists, for example Buhl, contend that such a transition is impossible. But when the disease has attacked the *upper lobes*, and the process of absorption has been imperfect, or considerable remnants have been left, which could be clearly recognized during life, we find that sooner or later consumption is developed, which sometimes runs a rapid, and at others a very gradual course. In such cases it may perhaps be allowable to say that the consumption would not have occurred but for the pneumonia of the upper lobes. In this locality pneumonia very often runs a more unfavorable course from the start, as compared with pneumonia of the lower lobes. The fever subsides at a later period, the dulness disappears more slowly, and convalescence progresses more unfavorably. But when pneumonia of the apex results in this way, how is it possible to decide whether there may not have been antecedent disease in the lungs or elsewhere to account for the abnormal character of the inflammatory products? The objection still remains, therefore, that in cases of croupous pneumonia terminating in phthisis, a result which clinical observation has shown to unquestionably occur, conditions may have previously been present which were favorable to the development of the latter. We cannot get rid of the clinical fact that pneumonia, so far as it affects the upper lobes, may end in pulmonary consumption, even in persons who must be regarded as perfectly healthy before the beginning of the disease, and it must therefore remain an open question whether,

notwithstanding the apparent soundness of the patient, there had not been previously existing foci of disease, such as caseous lymphatic glands, etc.

But if croupous pneumonia occur in a person who is already predisposed to consumption, or is scrofulous, or already consumptive, then even the inflammation in the lower lobes may terminate in gradual destruction of the lun with continuous fever and exhaustion of the vital forces.

Catarrhal pneumonia is regarded anatomically as well as clinically as a frequent forerunner of pulmonary consumption. From an anatomical standpoint this view may perhaps be justified, if most of the inflammatory parenchymatous changes found in the lungs of fatal cases of phthisis are to be regarded as catarrhal pneumonias, and if both the gelatinous infiltration of Laënnec and Rokitansky, and the tuberculous infiltration which they held resulted from the breaking down of the gelatinous infiltration into yellow (caseous) matter, are to be resolved into a mere modification of the inflammatory process; but from the clinical standpoint the connection referred to cannot be maintained. In all cases the exquisite forms of catarrhal pneumonia bear the same relation to consumption as the croupous variety.[1]

Catarrhal pneumonia is almost always situated first or solely in the lower lobes. Bartels and Ziemssen have each seen only two cases in which catarrhal pneumonia was followed by caseous products and miliary tubercles. The course of the disease is then always very rapid, as it was in the case recorded by Rilliet and Barthez,[2] who have also rarely seen pneumonic inflammations terminate in pulmonary phthisis. From these observations it is impossible to decide whether the caseous masses and tubercles found at the autopsy were the result of the pneumonia or had preceded it.

The belief that *pleurisy* is a cause of consumption seems to have been based upon the fact that in many cases of phthisis both the history and the physical examination show that pleurisy had previously occurred on the side in which the paren-

[1] *Bartels*, Virchow's Arch., XXI., S. 65.—*Ziemssen*, Pleuritis and Pneumonie im Kindesalter, S. 330.

[2] Rilliet et Barthez. Paris, 1843, T. III., p. 109.

chymatous changes are most strongly marked, or to which they were exclusively confined. It is a matter of common observation that sometimes when pleurisy occurs without any previous pulmonary symptoms, disease of the apex manifests itself subsequently, either in the farther progress of the pleurisy or a variable length of time after its disappearance. Furthermore, considerable remains of pleurisy are very often found at the autopsies of consumptives at parts of the chest (lower half) where there are no signs of phthisis, or only indications of its early stages, showing that the pleurisy is apparently not the result of the degeneration of the lungs.

But these observations are not sufficient to warrant the assertion that pleurisy or its remnants can produce a disease of the lungs. Here also it is always a pertinent objection that foci were already present in the lungs at the time the pleurisy took place, and that even if they were not the sole cause of the pleurisy, they at least favored its occurrence, and made the exudation more abundant and of worse quality. The influence of pleuritic exudations upon pulmonary phthisis amounts, therefore, essentially to this: that, when they last for a considerable time, general and local conditions of an unfavorable character, such as fever, anæmia, and imperfect expansion of the chest, are developed, conditions which also in other ways often lead to the development of phthisis; or these exudations are merely a complication of already existing pulmonary disease, and hasten its more rapid development into phthisis.

This effect of pleurisy is noticed especially when the exudation continues for a long time, and is imperfectly absorbed; while the more acute the attack and the more rapidly its products disappear, the rarer is the occurrence of such a result. These latter cases of favorable pleurisy are met with in persons of general good health and strong constitutions, while the chronic forms usually occur in those who are already predisposed in other ways to phthisis. Pleurisy may therefore be classed among the exciting causes, which, in the presence of an already exciting diathesis, bring to a more rapid development the perhaps hitherto latent phthisis.

Pulmonary hemorrhage, or, as we say, *bronchial hemor-*

rhage, was regarded by the ancients as a cause of consumption. Recently this question has been revived, and its importance insisted upon (Niemeyer).[1] Hæmoptysis has been recognized by physicians in all ages as a very common symptom of consumption, and there are numerous cases in which it seems to be the *first indication of disease*. That this fact is insufficient to stamp the hemorrhage as the cause of the pulmonary affection, which does not manifest itself until later, is evident from the reasons already repeatedly stated. Antecedent lesions may very well be present, notwithstanding our inability to detect them. Even foci of disease of very considerable size may escape our notice if they are distant from the surface of the lungs, and separated from it by tissue which contains air; and still more frequently the subjective symptoms may be absent, even when the organic affection has reached a considerable degree of development. As to the view that inflammation and caseous changes can be excited by the irritation from decomposing blood retained in the bronchi, such a result is to be regarded as exceptional. Niemeyer[2] records an instance of this kind, which is interesting on account of its very rarity, but it does not prove that the occurrence of fever on the second or third day after an hæmoptysis, or the increase of an already existing fever, is due to pneumonic processes resulting from the decomposition of retained blood. The new inflammation may depend upon the same cause which excited the hemorrhage. Moreover, as there is no evidence that the rupture of the vessel in these cases is due to a traumatic or any other known cause of like character, it is most natural to suppose that the wall of the vessel was already diseased, either from tuberculous deposit in it, or from a softening process extending to it from without. In fact, for aught we can say to the contrary, the bleeding may even have proceeded from a cavity, because when this is of small size and centrally situated, we have no means of discovering its existence. It is much more reasonable to suppose, moreover, that in every considerable hemorrhage the bleeding comes not from the capil-

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[1] *Niemeyer—Ott*, Klinische Vorträge über Lungenschwindsucht.

[2] L. c., pp. 53–59.

laries, but from the rupture of a vessel of some size. In what proportion of cases the bronchial mucous membrane should be regarded as the source of the hæmoptysis, we have no means of determining. The very interesting case of laryngitis hæmorrhagica published by Fränkel[1] is exceptional in character, and is therefore not available for the explanation of the ordinary forms of bronchial hemorrhage. On the other hand, experiments upon animals and observations in the human subject have repeatedly shown that blood extravasated into the air-passages is readily absorbed without doing any harm, and that neither traumatic hemorrhages in these parts, nor the hemorrhages caused by heart disease are capable of developing phthisis or even only pneumonic processes. But when blood is retained in parts already diseased, and there putrefies in consequence of its failure to be absorbed or removed in any other way, it may then excite inflammation, and in this sense pulmonary hemorrhage may hasten the phthisical process.

For these reasons it seems to me that the judicious remarks of Louis[2] on this point are still true at the present day: "Rien n'est mieux prouvé aujourd'hui, pour tous les médecins observateurs, que l'extrême rareté des hémoptysies de quelque gravité, indépendantes des tubercules; de telle sorte, qu'en admettant, pour un moment, que de pareilles hémoptysies soient quelquefois la cause excitante d'une éruption de tubercules cela ne pourrait être démontré. Il est donc impossible, dans l'etat actuel des choses, de régarder l'hémoptysie, forte ou faible, comme une cause des tubercules."

Besides the morbid changes in the respiratory organs, there are also certain general diseases which lead to phthisis. This disease not infreqently occurs in persons who have had an acute attack of fever, or who are still suffering from chronic disease. In many of these cases the explanation of such a sequence, or it may be casual connection, lies in the conditions already discussed.

Long-continued fever induces anæmia, and leaves behind it

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[1] Berliner klinische Wochenschrift, 1874, No. 2.

[2] Recherches sur la phthisie. Paris, 1843, p. 608.

such a deranged nutrition of the tissues that caseous processes may either be developed from existing germs or residues of disease, or may be excited by slight external causes. In this way any serious disease or difficult convalescence may lead to consumption. This connection seems to be more direct when the respiratory organs have been the seat of morbid change during the course of the febrile disease, as is the case especially in the long-continued fever of typhoid affections. Bronchial catarrh is always present in these diseases, and very often also in the upper lobes. Moreover, atelectases frequently take place in consequence of the feeble breathing resulting from muscular weakness; and the inspissated secretion in the bronchus leading to the atelectatic part may excite bronchial inflammation followed by pneumonic processes, ulcerative destructions, and other early lesions of phthisis, which may go on to further development, especially in those who under favorable circumstances are unable to effect a restoration of their wasted tissues.

The connection between *measles* and consumption is generally acknowledged. The influence of measles is manifested in various ways. In the first place bronchial catarrh is always present, and very often, associated with it, disease of the bronchial glands. Other complications, also, frequently arise in the shape of atelectasis, inflammatory processes in the parenchyma of the lungs, and occasionally acute miliary tuberculosis, which is observed more frequently after epidemics of measles than under any other circumstances. But simple bronchial catarrh and catarrhal pneumonia do not of themselves produce phthisis, and we must therefore infer a special cause for the alleged frequency of this disease after measles. The explanation must lie in the occurrence of scrofula, and in the infiltration and caseation of the lymphatic glands, which are so common after measles; and the supposition of this intermediate factor in the production of phthisis is supported by the frequency with which acute miliary tuberculosis occurs in this connection.

The frequent occurrence of pulmonary consumption after *pertussis* is probably to be explained in the same way. That this connection is by no means uncommon is beyond question, and, according to Rilliet and Barthez, disease of the bronchial

glands and lungs is almost exclusively the only lesion which has been observed to follow whooping-cough.

Consumption is also common in the later stages of *diabetes mellitus*. The explanation of this complication is at present not clear. The apex disease sometimes occurs before the patient has become much prostrated; but when the pulmonary affection has once made its appearance a more rapid marasmus usually sets in. The abnormal condition of nutrition in this disease is the only cause we can assign for the peculiar metamorphoses of morbid products. In fact, simple pneumonia in diabetes often terminates in gangrene.

In connection with these well-known examples of consumption being produced by antecedent diseases, it is proper to mention also the *puerperal condition*. Here, also, the anæmia and exhaustion, resulting from the lying-in, are not a sufficient explanation. In most of these cases the puerperal state has been preceded by manifest symptoms of apex disease, or by a pronounced disposition to phthisis. During pregnancy the incipient disease usually becomes stationary, but after the confinement it assumes a florid type, and sometimes results fatally within a few months. In some instances, however, the pulmonary symptoms do not manifest themselves until after the puerperal period. The more frequent the confinements, and the shorter the intervals between them, in such women, the more certainly does the disease become fatal. When the influences of lactation were under consideration it was mentioned that these rules are not without their exceptions. It remains only to add that the particular modes in which this period of regressive and revolutionary changes in the female organism seems to favor the occurrence and rapid extension of pulmonary consumption are at present unknown.

As regards the influence of *age*, acute miliary tuberculosis occurs occasionally in early infancy, but pulmonary phthisis is observed only after the first dentition, and the mortality from this cause in childhood reaches its maximum between the fourth and seventh years. Its actual maximum occurs between the fifteenth and twenty-fifth years; the mortality then remains high until the thirty-fifth year, and after this time steadily

sinks. The most common causes of phthisis among children, besides a strong predisposition, are scrofula, measles, pertussis, unfavorable vital conditions, bad air, and insufficient nourishment; hence the special prevalence of the disease among the children of the poor in large cities. At a later period other causes come into play in the production of apex disease, such as the imperfect development of the chest, and the position of the body in certain employments, co-operating with hereditary predisposition, anæmia, the bad effects of the occupation, etc.

Whether *sex* has any influence upon the occurrence of phthisis is doubtful. It would not be surprising if women were attacked in somewhat larger proportion, because they have less power of resistance; they are more subject to anæmia, and their sedentary mode of life interferes with the proper expansion of the chest; while they are equally exposed to other injurious influences, those connected with the occupation in males being counterbalanced in females by the effect of confinement and lactation. Still it seems to me that pathology has as a rule been very little advanced by calculations of this kind, and aside from the objectionable character of all such deductions, unless they are formed from the comparison of a very large number of cases, the discovery of a striking difference as regards age or sex would have no practical bearing except as it led to further investigations of the cause. The statistics of mortality from consumption which we already possess are certainly sufficiently large, but they do not present any such striking difference in regard to sex as to make it particularly important to have the question settled.

In the investigation of the causes of a disease it is of course important to know the conditions which prevent or seem to prevent its occurrence. Allusion has already been made to the fact that there are countries in which, on account of their elevated position, the dryness of the air, or the uniform temperature, this disease is uncommon, or does not occur at all. It is commonly supposed, also, that where malaria prevails phthisis is absent, or at least rare; but there are numerous observations to the contrary.[1] Both phthisis and malarial affections abound in the

[1] *Hirsch*, Geogr. Path. II., 99.

Netherlands, in many districts of France, in Alsace, Oldenburg, Hungary, South Russia, and even in Algiers, the very country with regard to which Boudin formulated the law of antagonism; while in other countries we see both diseases prevailing endemically alongside of each other. Moreover, the fact that intermittent fevers disappear after the ground has been drained, and are replaced by consumption, may perhaps be explained, as Hirsch has suggested, by the simultaneous changes in the social conditions of the inhabitants, by the increase in the density of the population, the establishment of manufactories, etc.; circumstances which everywhere favor the occurrence of phthisis.

The relation of *cancer* to phthisis is different. Although the antagonism between this disease and consumption has been disproved in numerous cases (in my government examination I had to work up the case of a man fifty-two years of age, in whom recent pulmonary and intestinal consumption co-existed with cancer of the liver and stomach), yet general experience shows that cancer is most frequent in organs where caseous masses are most uncommon; that in the lungs cancer is as rare as consumption is frequent; and that notwithstanding the vast number of cases of consumption it is rare to find one complicated with cancer. This fact, however, does not seem to have any bearing upon the explanation of the genesis of the two diseases. An article by Cooke[1] shows that a reaction is setting in against the doctrine of antagonism. This writer found that seventy-nine women with carcinoma mammæ had thirty-seven tuberculous children, and he regards this fact as an evidence of relationship between cancer and tuberculosis.

A few other conditions remain to be considered, or rather, perhaps, to be still further investigated.

In the first place *goître*, and *emphysema* are said to be antagonistic to phthisis, and the same is maintained regarding *valvular diseases of the heart* and a considerable degree of *contraction of the chest*, such as is produced by spinal curvatures.

To each of these rules there are, of course, a few exceptions, but the correctness of the general statements must probably be admitted.

[1] Med. Times, 1867, p. 538.

Among the valvular affections, mitral stenosis is probably the one most rarely complicated by pulmonary consumption; in lesions of the aortic valves phthisis is less infrequent. In the case of mitral stenosis the antagonism may perhaps be explained, in accordance with Traube's view,[1] by the more copious effusion from the pulmonary vessels, whereby the desiccation and caseation of morbid products are prevented. The same explanation may answer also for scoliosis, but not for struma and emphysema, at least not directly. In all of these pathological conditions there is hypertrophy of the right ventricle, with an increased activity of the respiratory muscles. But I must content myself with merely alluding to this subject, because a discussion of hypotheses is out of place here.

PATHOLOGY.

General Description and Course of the Disease.

Pulmonary consumption is essentially a chronic disease. Even the acute form, known as phthisis florida, generally requires several months to complete its course. Acute miliary tuberculosis is so different in its general picture from all forms of consumption that it ought to occupy a position by itself. The early symptoms of phthisis are generally so indefinite that even the most careful examination will fail at the outset to demonstrate the existence of the disease, and the diagnosis is based rather upon apprehension than knowledge. Moreover, the statements of the patient himself, even if he be a physician, are usually entirely untrustworthy. According to the degree of education (which is not to be confounded with the social position) the beginning of the disease is sometimes referred to a particular event, and at other times is left quite indefinite. Each patient has his own notion as to the cause of all the ills of the world; some ascribe them to worry, others to anxiety or fright, or to catching cold, or to cold drinks; sometimes these are all summed up in a common cause, as, for instance, a war, and then

[1] Bemerkungen über das Verhältniss der tuberculösen Pneumonie zu den organischen Herzkrankheiten. Med. Centralzeitung, Dec., 1864.

for years afterwards everything is referred "to the campaign." Of such an event the patient has a lively recollection, but the causes, which were in operation before that time, escape his memory, being obliterated by the more vivid impression. It should also be borne in mind that at the outset of the disease the symptoms usually intermit or remit for a considerable time, and that we may be unable to learn anything of these earlier periods on subsequent examination. Or perhaps others may have engraved upon the empty tablet of the patient's memory an artificial anamnesis, such as we find in the books. Another no less important reason why these early symptoms are overlooked is the well-known hopefulness of consumptives, which leads them to take life easily, and to think of everything else rather than the gravity of their complaint, thus preventing their giving any intelligent attention to their own condition. In fact this characteristic peculiarity, which is almost diagnostic, and which never leaves the patient until his last breath, but rather increases with the progress of the disease, is shared also by his relations, and for this reason even their statements are usually not to be accepted.

Generally speaking, two classes of cases are to be distinguished. In one class, for some time before the beginning of a single symptom, indications of a general derangement of nutrition are noticed, such as pallor, weakness, loss of appetite, and perhaps also more or less fever, lasting for weeks or months before cough or hæmoptysis, for instance, occurs ; in the other class the usual symptoms of the incipient disease make their appearance, and continue without any evidence of other disturbance of health, and without attracting much attention, until at last muscular weakness, pallor, emaciation, and febrile symptoms set in, with night-sweats, which more than any other symptom attract the attention of the patient.

In many of these cases where an indifferent state of health continues for a long time without any chest symptoms, or perhaps even without any past history of cough, an examination will show that lesions already exist in the apices. Or febrile symptoms may be detected by the thermometer, and excite suspicion of phthisis, if no cause can be found for them elsewhere in the body, or in the vital conditions of the patient. Among

the latent incipient symptoms we must also include a special excitability of the heart in anæmic persons, a continuous acceleration of the pulse without any evidence of increased temperature. In rarer instances the pulmonary affection is disguised by the presence of digestive derangements in the stomach or intestinal canal, lasting for a considerable time before the respiratory organs are affected. By the older writers dyspepsia was regarded as a very important factor in the causation of phthisis. Obstinate diarrhœa has already been mentioned among the causes of the disease, although it is still doubtful whether the bowel affection depends from the start upon tuberculous processes.

Generally, however, we are obliged to date the beginning of the disease from a cough, which arises without obvious cause, and either with or without a special disposition to phthisis; in other cases the latent morbid process seems to manifest itself first in an hæmoptysis, which is followed immediately, or after a considerable interval, by cough. On further examination of such patients it will very generally be found that their parents or grandparents have been consumptives, or that some of their aunts or uncles, or their own brothers and sisters have died of some form of phthisis (including meningeal tuberculosis); or else that in their childhood they had been scrofulous, or had had pneumonia or pleurisy, or had been exposed to unfavorable conditions of life, or to some of the causes mentioned in the previous chapter.

If such a person have large eyes, with a transparent sclerotic and long eyelids, a pale face which blushes readily, thin, slender hands, with incurvated nails, and a sharply defined red line at the edge of the gums, opposite the incisor and canine teeth, the experienced physician will at first sight suspect the existence of phthisis. When the clothing is removed, one is struck with the leanness of the chest, the hollowness of the subclavicular depression, and of the supra- and infraspinatous fossæ, the prominence of the scapulæ, the small antero-posterior diameter of the thorax, the width of the intercostal spaces, and the abdominal character of the respiratory movements. The upper parts of the chest, on both sides, or on one side particularly, move but very slightly when the patient voluntarily takes deep breaths;

the number of respirations is somewhat increased, the action of the heart irritable, and the pulse accelerated. Upon the skin of the breast and back are found several yellowish spots, which desquamate slightly (pityriasis versicolor). The temperature in the morning is normal or subnormal, but during the afternoon or evening it rises a little (100.4° F. in the axilla), the cheeks become flushed, and the hands hot. The patient says that within the last few weeks she has lost more or less flesh, and has had a slight cough of a dry, hacking character, and sometimes accompanied by pain in the region of the shoulder. This pain is also present at other times to a moderate extent. All the other functions are undisturbed, except that the last menstruation was more scanty than usual.

The examination of the upper parts of the lungs shows already on one side prolonged, harsh expiration, and on coughing a few crackling râles, which are not heard in any other part of the chest. The percussion note is somewhat shorter here than over the corresponding region on the other side. At present nothing abnormal is found in the other organs.

If the patient faithfully carry out the directions laid down, her condition improves after a few weeks, she gains in strength and weight, and the evening rise in temperature is no longer noticed. For some time everything seems favorable, only the cough does not entirely disappear, and some shortness of breath shows that her arch-enemy still exists. Then, perhaps in March, the symptoms begin again to grow worse, the cough becomes stronger, and suddenly an hæmoptysis occurs, with a sharp, irritable cough, and some ounces of bright red blood are lost during the course of a few hours. Fever again sets in even during the morning hours, slight perspirations take place at night, the crackling râles in the affected part of the upper lobe become more abundant, and at times present a sibilant character; the expiratory murmur, especially in the supra-spinous fossa, is sharply bronchial, and the dulness unmistakable. The patient is obliged to keep her bed, and loses flesh perceptibly. The expectoration now consists of small yellowish masses, containing white streaks, and granules of more solid consistence, which gradually sink in water. In many cases microscopical examina-

tion reveals distinct elastic fibres. The appetite is somewhat diminished and the pulse accelerated.

But from this relapse also the patient once more recovers, and after leading for some time an out-door life, with proper care as to diet, she seems to have regained all the ground she had lost. The dulness and the bronchial breathing, however, still remain, and the crackling râles having almost disappeared, the bronchial breathing may now be heard also in inspiration.

The hopes of the physician and of the friends again revive; the patient herself has never lost hers. But disappointment again returns, because in the autumn the cough and the expectoration once more increase; stabbing pains repeatedly occur in the side originally affected; a transient friction is heard in the axillary line; the dulness in the upper parts becomes more extensive and is noticeable also upon the other side. On auscultation abundant crackling râles are again heard, and the bronchial breathing gradually acquires an amphoric quality, while the percussion sound becomes clearer again and somewhat tympanitic. The fever also reappears, and after this time maintains its ground. Sometimes the thermometer does not rise above 101° F. for several days; at other times it reaches 103.3° F., while in the morning the temperature is not above the normal. The patient's strength is obviously impaired, although there is no anorexia, and the alvine discharges, as a rule, remain normal. The emaciation gradually becomes extreme; the expectoration increases, and contains solid conglobate, irregularly spherical sputa, which sink to the bottom of water, and at the same time others which are more fluid and entirely purulent. The night-sweats become more profuse, and the physician now sees that he has an invincible foe to combat. The patient looks forward to complete recovery in the spring, when she intends to visit a watering-place, but instead of this she sinks into a state of extreme exhaustion, and death gently closes the scene.

Cases of this kind represent the simplest form of pulmonary consumption, but they are by no means the most common. The symptoms and the course of the disease are modified in various ways by the usual extension of the process to other organs or by complications. The most frequent of these are symptoms on

the part of the larynx and intestines. Sometimes in the earlier stages, at other times later in the disease, the patient becomes hoarse, and complains of a slight burning sensation in the larynx. The cough excites some pain at that point, and deglutition may become difficult. The hoarseness gradually increases to aphonia; and, on drinking, some of the fluid may enter the larynx and excite severe paroxysms of coughing; sometimes signs of laryngeal stenosis occur, though only temporarily. These symptoms are explained by the laryngoscopical lesions, which consist of a variable amount of inflammation, generally in the posterior wall, and extending thence anteriorly along the vocal cords. Yellowish nodules are also found, which present indications of ulcerative destruction; the small ulcers unite to form larger ones, and sometimes on the arytenoid cartilages are to be seen œdematous swellings, which may disappear after a time. For the development of this degree of laryngeal disease more or less time may be necessary. Usually the laryngeal symptoms progress slowly, and the ulcerations are generally so situated that the actual suffering is inconsiderable, the chief inconvenience being the increasing hoarseness, which persists to the end.

The intestinal affection also makes its first appearance in the later course of the pulmonary disease, except in those rare cases, previously mentioned, in which the phthisical nature of the bowel disturbance remains doubtful because no lesions can be detected in the lungs. The intestinal lesions are generally at first situated above the ileo-cæcal valve, and hence the only symptoms noticed are a moderate increase in frequency and looseness of the discharges, and slight pain upon pressure in the region of the cæcum. These diarrhœas may be relieved by diet and medicines, but usually do not cease entirely. Even when there are only one or two discharges daily, they are still abnormal, and every now and then aggravations occur without any apparent cause, until at last even the most approved remedies fail to have any effect; the number of evacuations increases to six or eight, or more, in the twenty-four hours, and slight traces of blood and yellow specks of pus may be found in the stools. When the intestinal affection has advanced to this

degree the middle portion of the abdomen becomes slightly tympanitic, more resistant, and painful on pressure.

This frequent extension of the disease always results in rapid emaciation and loss of strength, even when the fever is not specially intense, and the rapidity with which the fatal result ensues depends directly upon the severity of the intestinal affection.

In rare instances the intestinal disease presents longer intervals of apparent or even actual arrest; in fact, even the severe and long-continued diarrhœa, which to all appearance is dependent upon tuberculosis of the bowels, sometimes ceases for a time, the patient recovers from his extreme prostration, and months elapse before a fresh attack of the disease occurs.

In the previous general description mention was made of the periods of arrest which usually occur during the course of phthisis. These periods may last not only for months but even for years, and in some cases, especially if the disease be confined to the lungs, the morbid process may terminate in a cure by cicatrization even after the formation of cavities, as has been repeatedly demonstrated at autopsies. Life may thus be prolonged for a considerable time, and in this sense we may speak of a permanent cure of phthisis. It was only last year that I made an autopsy at the clinic in this city on a woman ninety years of age, who had cavities in both upper lobes as large as a pigeon's egg, and extensively surrounded by slate-colored connective tissue.

Perforation of the pleura or of the intestinal peritoneum, and the complications with other diseases, by which the chronic course of phthisis is interrupted and the fatal event unexpectedly hastened, will be considered subsequently.

The special variety of the disease known as *phthisis florida* still requires to be mentioned. It runs its course within a few months, even when, as is not infrequently the case, the disease is confined exclusively to the lungs. The longer or shorter amendments, the temporary disappearances of the fever, the increase and decrease in the intensity of all the symptoms, which are the rule in the previously described chronic cases, are here entirely absent or are noticed in a very slight degree. The

course of this disease is more continuous and more constantly attended by fever, which is very often of a high grade; the marasmus is more rapid, and death is the speedy result.

In these cases no miliary tubercles are found at the autopsy; the only lesions are infiltrations, caseations, and excavations of such dimensions as to excite surprise on section of the lungs.

It cannot be denied, however, that improvement and arrest of the disease may occur in cases which are clearly of a florid type. In the Greifswald clinic I took charge of such a case after it had been treated by my predecessor, Niemeyer. It had been correctly diagnosticated as phthisis florida; in fact, it seemed as if the girl had only a short time to live, but the fever subsided, the expectoration diminished, the strength revived, the abnormal auscultatory and percussion sounds in the left upper lobe gradually disappeared with the exception of the signs of cavities, and her nutrition improved so much that she was able to leave the hospital. A year afterwards a new attack of phthisis occurred after her confinement, and she died with intestinal symptoms.

While in many of these florid forms the cough is severe from the start, and exhausts the patient by the fatigue, loss of sleep, and profuse perspirations accompanying it, there are other cases in which the cough is not a very prominent symptom. The expectoration may also vary considerably in amount. This difference obviously depends upon the variable degree in which the mucous membrane of the larger bronchi participates in the morbid process. Sometimes this membrane is actively hyperæmic and secretes freely, and then there will be considerable cough and expectoration; but if the lesions be confined to the terminal bronchi and the alveoli this will not be the case.

Other pulmonary symptoms now occur; the breathing becomes more rapid, and a sense of oppression or pleuritic pains compels the patient to occupy certain positions, in which these discomforts are least felt. He complains chiefly of increasing weakness, which he ascribes to his profuse perspirations. The fever is, however, the most important symptom. It is continuous, but varies considerably in intensity in different cases. In many cases the variation between the morning and the evening

temperatures is not greater than it is in typhus; for weeks together it never falls below 102.2° F., and often rises above 104° F. And yet, notwithstanding the continuous high temperature, typhoid symptoms, delirium, or stupor do not occur at all, or only to a very slight degree; a point to which I wish to call special attention. In other cases the fever curve presents a more marked downfall in the morning, while in occasional instances the highest point is reached in the forenoon. But, however much the fever may vary in these respects, either in different cases or at different times in the same case, it always remains the most conspicuous symptom, and is undoubtedly the true cause of the rapid emaciation, though, of course, much of the danger to life must come from the rapidly increasing disorganization of all the vital organs. The high temperature is always accompanied by a considerable increase in the frequency of the contractions of the heart. The pulse rarely falls below 120, and often rises above 130.

In this form of consumption, just as in the usual remitting and intermitting forms, there also occur in other organs, the larynx and intestines, anatomical lesions, and the functional derangements dependent upon them; but these rarely have time to develop to any considerable extent, and consequently the hoarseness and diarrhœa are not very troublesome.

The florid form of phthisis is most common in young persons; and as far as the period of life preceding puberty is concerned, it may be laid down as a rule that the younger the individual when the disease begins, the more rapid is its course. The special causes of phthisis florida are, however, unknown. It seems to be as prevalent among the better classes as among the poorer population; and the influence of such causes as hereditary predisposition, the conformation of the chest, and an intense scrofulous diathesis, does not appear to be greater in this variety of phthisis than in the chronic forms. The etiology of this affection requires farther investigation.

The last case of acute phthisis which I have seen was a young man, who had rapidly grown very tall, and had for years suffered from insufficiency of the aortic valves, and slight insufficiency of the bicuspid valves. The symptoms of phthisis devel-

oped while he was in the hospital; there were intense fever and profuse perspirations, which were entirely uncontrolled by remedies, and death ensued in the course of five weeks. The lesions in both lungs consisted of very extensive caseous destruction, with numerous recent bronchiectases lined with caseous matter, and a very few granulations in the infiltrated tissue, which might be regarded as miliary tubercles.

Analysis of Individual Symptoms.

The most constant symptom of pulmonary consumption is, of course, the *cough*. Although it varies considerably in severity, it is never entirely absent. To be sure we hear persons with evident lesions in the upper lobes of the lungs assert that they have had no cough, or only a slight hacking or clearing of the throat, or that they expectorate without coughing; but there is so much uncertainty about these cases, and they are comparatively so rare, that they do not disprove the rule that there is no consumption without cough. And yet it must be admitted that the pulmonary lesions, which lead to the development of consumption, occasionally continue for some time, and reach a certain degree of intensity, perhaps sufficient to be recognized, before the cough sets in; and to this extent it may be said that the cough is not always the criterion of the beginning of the disease. But these cases form only a small minority; or it may be impossible subsequently to determine whether the cough was coincident with the outset of the disease. As a rule, however, consumption begins with a cough. But it might be objected that very often the cough originally depended upon an ordinary catarrh, out of which the consumption developed subsequently; that the cough really antedates the latter, and in fact that this is the usual history. In my opinion this discussion is futile, because it is impossible to decide the question, and I therefore refer to what has already been said in reference to it under the head of etiology.

Once present, the cough continues to the end. Its severity, as a rule, runs parallel to the course of the disease. When the latter declines, or is arrested, the cough also subsides, and

increases again whenever the morbid process in the lungs takes a fresh start. The following points are to be considered in this connection.

In the first place, it is very probable that cough is excited the more easily the nearer to the larynx its cause is situated; and that when the irritation lies in the terminal bronchi, or in the pulmonary parenchyma itself, cough is less readily produced; consequently if the pulmonary lesion be slight or be developed very slowly, the cough may be absent. This fact would seem to confirm the supposition that the cough in consumption is only the expression of the implication of the bronchial mucous membrane, and that its severity does not always correspond to the changes in the parenchyma proper. Then again the implication of the bronchial mucous membrane may be merely accidental; it is an ordinary occurrence for a consumptive to add to his cough by catching cold. Or the true specific disease may extend upwards into the bronchi, and excite active reflex movements (cough) in certain cases, where the lesions in the parenchyma proper are less serious than in other persons, who have much less cough but much more advanced pulmonary changes. It should not be forgotten, also, that cough may be excited by morbid changes in the lymphatic glands situated near the bronchi and trachea, and that the extent to which they share in the phthisical processes must be taken into account in the causation of the cough. Finally, the occurrence of every reflex movement depends upon the condition of the nervous system. The more irritable the nervous centre, the more easily excited and the more powerful is the reflex action. That this is the case also with regard to cough is a matter of daily observation. Young persons, women, and other consumptives of excitable temperament have relatively the most severe cough, and at times their cough increases independently of any aggravation of the disease.

It appears, therefore, that certain exceptions will have to be made to the general statement above, that the severity of the cough is proportioned to the extent of the disease.

But, although the cough is a constant symptom, and as a rule increases and decreases with the phases of the disease,

the greatest contrast may of course be observed in different patients, and at different times in the same patient. While in many cases there is only a trifling cough up to the day of death, in others it is the chief source of annoyance throughout the whole disease. These violent efforts exhaust the invalid, induce perspiration, disturb sleep, and excite vomiting; they also inflict direct injury, and, unless moderated by treatment, they may hasten the course of the disease. But usually the cough of consumption is not, or at least only temporarily, specially severe or frequent. It is often excited more actively at certain times in the day, usually in the morning after sleep and at evening shortly after lying down. The following considerations will probably serve as an explanation. During sleep reflex action takes place with difficulty, and a greater accumulation of the irritant is necessary to excite it. The irritant in this case is the secretion, which goes on accumulating undisturbed during sleep until finally it becomes sufficient to induce cough, notwithstanding the sleep, and the patient is awakened; or the sleep is disturbed by some other cause, and then the increased excitability of the medulla produces a cough, which, on account of the accumulation of the irritation, lasts for a long time, until the cause is removed. The paroxysms of coughing which occur soon after lying down may be explained partly by the fact that, especially in morbidly hyperæmic tissues, the quantity of blood changes according to the position, and increases in the more dependent parts. The upper parts of the chest, and, of course, the large bronchi and the trachea, must receive more blood in the horizontal than in the upright position. Probably another cause of the increased cough is the difficulty in the removal of the secretion when the patient is lying on his side or back. We often see this fact illustrated by the patient's beginning to cough when he lies upon the side most diseased, the very position in which the conditions are most unfavorable for the discharge of the secretion, and most favorable for an increase of the hyperæmia.

At the beginning of the disease the cough is often inconsiderable, consisting only of a slight and infrequent hacking; but, in other cases, it may be severe and exhausting from the start.

These differences have already been explained. Towards the end the cough sometimes gradually declines. This disappearance of the chest symptoms is seen also upon the supervention of active intestinal disease, and is then probably due in part to the general exhaustion of the patient and to the remedies used.

There is probably no truth in the belief that there is a peculiar *timbre* and character to the cough of consumption which are pathognomonic of the disease. The timbre is affected by the condition of the vocal organs, and changes its character when these, and especially the larynx, become affected. The cough of phthisis may therefore present all gradations in intensity of sound. There is one variety of cough which may be said to be to a certain extent characteristic, viz., that which occurs towards the end of the disease, when the vocal cords are ulcerated and there is great muscular weakness.

The *expectoration* varies extremely in different patients, and in the different stages of the disease. While many patients usually expectorate but very little, others often fill up large cups every day for a considerable period, and in this way lose a large quantity of corporeal substance.

In many cases the cough is at first unattended by expectoration. The long continuance of such a dry cough has always been regarded as suspicious, because in an ordinary catarrh secretion and expectoration occur very early. But there are also many cases of phthisis, in which expectoration takes place from the start. In the former case we may suppose that the anatomical changes are at first extra-bronchial, and that there is no catarrh of the mucous membrane, while in the latter case the opposite is true. The sputa of the early part of the disease usually have no distinctive character, except when there has been considerable pneumonic infiltration, and then they have the same vitriform glutinous appearance which characterizes those of pneumonia independently of their color. The color is, however, less intense than that of the pneumonic sputa, and is at most only a slightly grayish red, which depends, of course, upon an admixture of red blood-corpuscles. Sometimes the expectoration presents still another character; the catarrhal sputa are mixed with blood, which can be distinctly recognized

in the form of dots and streaks, and this appearance is noticed especially in cases in which long continued and severe paroxysms of coughing occur. These vitriform sputa, which are sometimes scattered through the whole mass of the grayish-yellow expectorated mass in such small numbers as to be easily overlooked, are found, on examination, to contain large roundish cells, which are very granular in appearance, and are probably alveolar epithelial cells, which have undergone fatty degeneration. In the cadaver they may be seen with the naked eye as fine yellowish spots in the midst of the gray, transparent infiltration.[1] These vitriform sputa, containing the granular cells alluded to, indicate, in my opinion, the presence of a recent local pneumonia.

Sometimes even at an early period of the disease, but in many cases only in its subsequent progress, the sputa contain grayish white, opaque striæ and granules, which are usually seen only by transmitted light. The granules generally sink to the bottom of water, but the striæ, which present, moreover, a branching appearance, remain suspended on account of their intimate mixture with the rest of the yellow viscid mass. Bayle[2] has insisted upon the significance of these granules, and Louis upon that of the striated sputa. On microscopic examination these places are found to be distinguished from the rest of the expectoration by their containing a larger quantity of amorphous matter, and fewer cellular structures. By this time, and also in the farther course of the disease, we may often succeed in demonstrating the presence of elastic fibres of the lung tissue. According to Fenwick[3] the best way to find them is to mix the sputum with an equal quantity of a solution of caustic soda in distilled water (18 : 100), boil the mixture with frequent stirring, then add three or four times its bulk of water, and allow it to stand in a conical glass. The deposit contains the elastic fibres.

Gradually the sputa become globular and compact, their surface uneven, and the edges somewhat ragged, as if eroded; they fall to the bottom of the water in the vessel, or hang attached to

[1] *Virchow*, V.'s Arch., Bd. 1, S. 146.

[2] Recherches sur la phthisie pulmonaire. Paris, 1810, p. 25.

[3] Lancet, Dec., 1868.

a thread of mucus. They contain very little or no air, and look as if they had remained for a long time in one place, of which they had formed, as it were, a cast; in fact they seem to have come from small cavities. These cavities, which are found even at this early stage, consist at first of bronchiectases, which afterwards ulcerate, and are generally filled with a viscid, slightly granular, yellow substance, which is the specific product of the diseased mucous membrane. In these globular sputa also, the larger as well as the smaller, a considerable amount of amorphous granular matter is found in addition to the cellular elements. The older observers have also frequently described the expectoration of white masses of a more solid, mortar-like consistence, and looking like concretions; and accordingly they have even set up a special variety of the disease under the name of calculous phthisis. Portal[1] gives a detailed account of the expectoration of these stony bodies. In some of his cases there had been no indication whatever of phthisis, in others there were marked symptoms, which in some instances disappeared after the discharge of these substances. At other times this event was followed by the development of consumption either with or without hæmoptysis. These concretions are sometimes as large as a pea. Even as far back as the time of Morgagni[2] the occurrence was regarded as a very bad omen. Portal refers the origin of these concretions partly to the inhalation of particles of various kinds of dusts. Such particles are, however, rarely found in the expectoration, although the occurrence of concretions of various sizes in the lungs is very common. They are almost always surrounded by a thick capsule of cicatricial tissue, which prevents their being discharged. In case, however, they are discharged, it must be because they were incompletely inclosed, and became loosened by the softening of the surrounding parts. Hence the expectoration of these substances will probably indicate a progressive softening, rather than a curative process. Moreover, it should not be forgotten that these concretions may arise in the bronchial glands, and after ulcerating their way through the

[1] Observations sur la phthisie pulmonaire, I., 482 et seq. Paris, 1809.

[2] Epist. XV.

bronchial wall, and becoming detached, may make their appearance in the expectoration. A case is recorded where a child was even choked to death by a concretion of this kind as large as a cherry; it became impacted in the larynx, through which it was too large to pass.[1]

As the disease advances the expectoration becomes more and more purulent, and in many cases the sputa consist almost wholly of fluid pus, which is discharged in large quantities, and readily flows down the sides of the glass vessel. Most of this pus comes from the walls of cavities. Sometimes it collects in them, and then is discharged from time to time with more severe paroxysms of coughing, while in the intervals the expectoration is but slight, and in many cases the cough almost disappears. The occurrence of these accumulations is favored by the muscular weakness of the patient, which increases towards the end of the disease. Moreover, the pus contained in the cavities may decompose and putrefy, and then the sputa become offensive, present a more greenish appearance, and contain numerous other organic bodies, together with fat crystals. Such a fluid has also a macerating and eroding effect upon the walls of the cavities. The cicatricial tissue surrounding the cavities very often contains a considerable amount of pigment, which becomes mixed with the pus, and gives the sputa a dirty gray or even blackish color. On the other hand, during the later stages of the disease, the pus from this source has sometimes a slightly reddish tinge. Louis says that he has noticed this appearance only during the last days of life.

At any period of the disease the expectoration may, of course, be altered in quantity and consistence by the admixture of serum, which probably not infrequently escapes from the parenchyma as a result of a local œdema. In many patients large quantities of saliva and buccal mucus become mixed with the expectoration, and make it more fluid and clearer. These different kinds of sputa can, however, always be discriminated by means of the admixtures, and usually, especially in the later stages, several of them are to be found at the same time and in the same individual.

[1] *Ruehle*, Kehlkopfkrankheiten.

The expectoration of *blood* is a very significant symptom. While in some cases there may be only a few streaks, which seem to be of no importance, and may come from a great variety of sources, in many other instances the quantity is more abundant. The discharge may take place either gradually, accompanied by the ordinary cough, which is no stronger nor more frequent than usual during the attack; such hemorrhages sometimes continuing for days and weeks, and generally indicating a pneumonic process; or the blood may be expectorated so rapidly and in such large quantities that death is the immediate result. In many of these cases, however, the fatal event may not be due directly to the loss of blood, but may be partly the effect of asphyxia from obstruction of the respiratory passages. Death from such a hemorrhage is, however, a rare occurrence, although hæmoptysis, in the sense of the rapid discharge of large quantities of blood, takes place in more than one-third of the cases. The blood is bright colored, more or less aerated, and is expectorated with paroxysms of coughing, which are frequently very violent, and expel the blood through the nose and mouth at the same time. Within half an hour more than a litre may escape in this way. Violent paroxysms of coughing are very apt to be followed by vomiting, and because this act occurs also in severe hæmoptysis patients sometimes speak of the hemorrhage as a vomiting of blood. In order to avoid mistake we should inquire in regard to the character of the expectoration which immediately follows the supposed hæmatemesis. When the blood comes from the air-passages, a portion of it always remains behind, and is gradually discharged by the succeeding cough; its color, therefore, becomes darker and darker, and at last is a dirty brownish red. In hæmatemesis the blood first escapes by a single act of vomiting, and then follow black-colored discharges from the bowels. If the physician can see the discharged blood or the patient, it is not easy to mistake the source of the hemorrhage.

Sometimes the hæmoptysis is preceded by symptoms of cardiac excitement, a feeling of oppressed breathing and increased warmth, and a sweetish taste. The latter symptom is still more frequently noticed when the patient is apprehensive of a return

of the hemorrhage, and in such cases the cavity of the mouth should be examined. The sweetish taste may then be found to proceed from the bleeding of the gums; and this discovery affords an explanation, not only for the traditional connection between this symptom and the occurrence of hæmoptysis, but also for the statements of patients that they had found their mouths full of blood in the morning, and for the spots of blood which they had been alarmed at finding on their pillows. Many persons make sucking motions of the mouth during sleep, especially if any irritation be present in the buccal cavity, and this explains the escape of bloody saliva. In most cases hæmoptysis occurs without any antecedent sensations, and the patient is taken by surprise.

Not infrequently, however, the hæmoptysis may be ascribed to some special cause, such as severe coughing, violent physical exertion, a cold bath, straining at stool, or to something else which increases the force of the heart's action, and suddenly heightens the blood pressure in the pulmonary circulation. There is also a traditional belief, handed down from ancient times, that pulmonary hemorrhage may be produced by the arrest of a menstrual or hemorrhoidal flux, or of bleeding in some other part of the body. The patient himself, in his desire to find some other than the true explanation of his symptoms, is very apt to ascribe them to hemorrhoids, which he declares have shifted to his chest. And as to women, what malady do they not trace to a non-appearance of menstruation! But while I fully acknowledge that both patients and physicians, as experience has amply shown, are apt to conceal their ignorance under the diagnosis of hemorrhoids and menstrual derangements, I am, on the other hand, far from denying any connection at all between the interruption of a regularly recurring bleeding in one place and its reappearance elsewhere. Evidence on this point is, of course, difficult to obtain, and I can only refer to the analogous occurrence of epistaxes, symptoms of cerebral hyperæmia, and excitement of the vascular organs in general under these circumstances, and to the cases in which a menstrual or hemorrhoidal flux seems to have been connected with pulmonary hemorrhage. For these reasons I am of the opinion that,

provided lesions of the respiratory organs are already present, an arrest of menstruation, occurring independently of pregnancy, fever, or a previous anæmia, may be an exciting cause of hæmoptysis, and that the same thing is true of bleeding from piles. In regard to the latter, I may add that pulmonary hemorrhages of this kind have occurred in several persons of my acquaintance at intervals of from four to six weeks, and were relieved by the regular application of a few leeches to the anus at the expected time.

According to Louis, women suffer from hæmoptysis more frequently than men in the proportion of three to two. Still, no conclusion can be drawn from this fact in regard to the influence of menstrual derangements upon this symptom.

Experience shows that hæmoptysis may occur at any period of the disease, from its outset to its termination, and either in small or in large quantities. Previous to the beginning of this century, when the pathology of the disease came to be based upon the pathologico-anatomical views of the French writers, and a new importance was attached to the autopsy as last chapter of the patient's history, in fact, even as far back as the time of Hippocrates, hæmoptysis was regarded as a cause of phthisis, simply because it was in many cases the first sign of the disease. I have already expressed my doubt of the occurrence of such a truly primary hemorrhage when the pulmonary tissue is healthy, and have insisted that I regarded this belief that the disease can begin in a spontaneous hæmoptysis as indefensible. It may be added, however, that not unfrequently the hæmoptysis seems to be preceded by only a very brief history of disease, the patient stating that his slight cough has lasted only a few weeks, and has been unattended by any constitutional disturbance. But with our present knowledge even such cases cannot shake the conclusion that a rapid discharge of large quantities of fresh blood with cough can come only from the rupture of a vessel of some size. Lesions of this kind, resulting from already advanced pulmonary disease, are not uncommon even in persons who call themselves perfectly healthy and are so regarded by others. I have elsewhere [1] recorded a case of imme-

[1] *Volkmann's* Sammlung klinischer Vorträge, No. 30.

diately fatal pulmonary hemorrhage, in which a cavity was found containing the ruptured vessel, and yet no previous symptoms of disease had been noticed. No satisfactory evidence has as yet been advanced that hæmoptysis can proceed from a so-called parenchymatous bleeding, and yet the perforation of the smaller vessels by miliary tubercles (see chapter on the pathological anatomy) shows that repeated slight bleedings may possibly occur in this way. Nor do I think we are justified in regarding the hemorrhage, at least when it is copious, as coming from the bronchi; I have never yet seen any cases of this kind, and doubt their occurrence.

In the later course of phthisis no one hesitates to refer the bleeding to a cavity, although the injured vessel is by no means always found when an autopsy is made soon after the occurrence of the hemorrhage. To be sure, a cavity whose size need not bear a direct relation to the quantity of blood discharged, is often seen filled with remains of blood, while nowhere else is anything like the same amount to be found. But the blood may disappear very rapidly even from cavities, as I have seen quite recently. In a patient with consumption who died only two days after the arrest of a copious hemorrhage, the numerous cavities of various sizes and all the bronchi were free from any bloody coloration. Hæmoptysis stands in no clear relation to the course of phthisis. It may be absent in the most rapid forms, and this is very frequently the case; or it may recur many times in the slowest, as is also not uncommon. In the latter case the patient finally comes to disregard his attacks; he becomes accustomed to them, as do also those about him, and the medical adviser is therefore not consulted. Here, also, the more severe hæmoptyses must be attributed exclusively to cavities. Patients may live to old age with such cavities, recovering with lungs essentially unimpaired from the successive hemorrhages, which only rarely exert any injurious influence upon the course of the disease, or endanger life by direct loss of blood. I have, however, already referred to the fact that the vascular rupture may be due to the occurrence of fresh processes in the neighborhood of the vessel, and that the new outbreak of the disease may thus date from the hemorrhage,

and perhaps also may be excited by the decomposition of retained blood.

At the outset of the disease, when reliable symptoms are wanting, hemorrhage is to be regarded as a very important symptom; in the later stages, especially in chronic cases, it may be comparatively unimportant; it is rarely dangerous to life.

While cough and expectoration are constant symptoms of consumption, and hæmoptysis is a common occurrence, the patient, on the other hand, suffers but little from *pains* in the chest; at least the more severe stabbing pains are exceptional, and undoubtedly depend upon complication with acute pleurisy, or perhaps pneumothorax. The rarity of pain is remarkable, in view of the fact that adhesions of the pleural surfaces, or some other product of pleural inflammation, are never absent. And yet it is undoubtedly true that the pleura, and not the pulmonary parenchyma itself, is the source of the pains which do occur in affections of the lungs. Even the most intense hyperæmia and inflammation, and even ulcerations in the bronchial mucous membrane, never excite actual pain. Yet at the autopsy in cases of consumption we always find evidences of previous pleuritis, notwithstanding the slight history of pain. This experience is, however, not confined to phthisis; we also very often meet with general adhesion of the pleural surfaces, and with various thickenings and opacities of the same in other affections, where there has been no complaint of pain in the chest during life, and the same thing occurs in the case of the peritoneum. On the other hand, some patients suffer from severe stabbing pains in the chest, and when pneumothorax takes place these are sometimes overwhelming.

The sensation of pain must, at all events, depend upon the rapidity with which the sensitive nerve fibre is excited, and upon the number of fibres affected. In most cases of phthisis, however, the pleuritis is chronic, and limited to places of small size, beyond which the extension is gradual. Moreover, it affects in the first place the apices, the very locality in which the irritated pleura is most protected, and the respiratory movements and cough are probably least likely to do harm by the displacement or tearing of the irritated tissue, in consequence

of the comparative immobility of these parts. In sensitive persons, however, who are more attentive to their feelings, complaints of pain are not infrequent, and probably always correspond to the situation of the pleuritis. Independently of the cases in which the pleuritis seems to be the precursor of the disease, producing sharp pains in the lateral and inferior parts of the affected side before any other symptoms of the pulmonary affection are noticed, the first sensations of pain are felt in the region of the clavicle and scapula, or between the scapulæ. Many patients describe them as shooting from behind forwards. They are increased by deep respirations, coughing, pressure, and percussion, but are not, as a rule, insupportable, and usually they do not interfere with the breathing.

At a later period pains occur further down, which are increased by the same causes, but in this case also no friction sound can be detected on auscultation.

If circumscribed pains of this character be increased merely by pressure upon the intercostal spaces, this fact is sufficient of itself to show that there is a localized pleuritis, and, in consumptives, also an affection of the parenchyma of the lungs, because in these patients pleuritis can be regarded only as secondary to parenchymatous disease. These pleuritic pains are, moreover, apt to be mistaken for those of intercostal neuralgia, which also occurs in phthisis, and in some cases they are regarded by both patients and physicians as rheumatic, because they frequently change their position, and, when situated in the region of the shoulder, are increased by movements of the arm.

If the course of the disease be interrupted by an acute and more extensive pleurisy, the pains in the chest become more severe, and most intense when the pleurisy is excited by the occurrence of pneumothorax. In the latter cases the pain begins suddenly, and extends over a large portion of one side of the chest. The suffering is so great that the patient apprehends suffocation. After its first intensity has subsided, the pain still continues for some time so severe that he is unable to lie on the affected side.

Like the violent pain, *dyspnœa* is also one of the less common symptoms of phthisis.

In phthisis, as in other affections, we must distinguish between dyspnœa and a mere increase in the number of respirations, because these two symptoms are dependent upon different causes, and are not always excited simultaneously. But here we shall have to consider them together.

In the usual course of the disease these symptoms are noticed only to a moderate degree, although the pulmonary affection may be quite extensive. But if the frequency of the respirations, so far as it is determined by the lungs, depends upon the irritation of the extremities of the vagus, and if the dyspnœa is directly proportioned to the diminution of the respiratory surface (Traube), we ought to expect these symptoms to be much more urgent. The explanation may lie in the gradual manner in which the local changes take place. The functional disturbances which arise in the body from a given lesion seem to be less marked the more slowly the latter is developed. For example, when a pleuritic effusion forms gradually, and finally comes to fill one side of the chest, compress the lung completely and diminish the respiratory surface by probably one-half, there is but little dyspnœa and but a slight increase in the number of respirations, while both of these symptoms are very marked when a pneumothorax suddenly occurs. But in phthisis this comparative freedom from dyspnœa is noticeable only so long as active exercise is avoided. When the patient keeps perfectly quiet in bed or in his chair the respirations are but slightly more frequent than normal, even in the later stages of the disease, and the scaleni can scarcely be felt to contract on inspiration. But as soon as any exertion is required, the breathing becomes more difficult, and the patient complains of oppression over the sternum. Especially after repeated paroxysms of coughing, or on going up stairs, the respirations become more frequent and urgent.

But as soon as some intercurrent affection of the respiratory organs occurs this state of things is changed.

If the bronchi become filled with blood or serum, or if pleuritis, pneumothorax, œdema of the lungs, or a copious eruption of miliary tubercles take place, the breathing often becomes more rapid, and the dyspnœa severe. This result is especially

apt to be produced by miliary tubercles, which act as numerous points of irritation for the extremities of the vagus, and yet diminish the respiratory surface to only a very slight extent.

Fever is also another cause for increased frequency of respiration. In excitable persons, especially in children, the height of the fever may be approximately inferred from the number of respirations during sleep. Consumptives who have much fever breathe more rapidly than normal, and in many cases the respirations are for this reason more frequent in the evening than in the morning.

In children dyspnœa, which is strikingly periodical in character and is associated with a harsh ringing cough occurring in violent paroxysms, may be produced by the pressure of enlarged bronchial glands upon the nerves and bronchi.

Inspection of the Thorax.

When the etiology of the disease was under consideration, allusion was made to the causation of phthisis by an abnormal configuration of the thorax, and to the investigations of Freund, which show that in young children anomalies of the first ribs may produce a narrowing of the upper portion of the chest, and thus prove injurious to the apices of the lungs lying behind these parts.

In all times it has been noticed that striking *alterations* in the *form* of the chest were especially common in families with a strong hereditary predisposition to phthisis, as well as in patients actually suffering from the disease. These alterations have become familiar to us under the term "*paralytic thorax*," the chief characteristics of which are that the chest is flattened, and not infrequently elongated in its vertical dimensions.

The most striking feature is the small sterno-vertebral diameter. This is so marked that it can be detected by merely looking at the profile of the chest. As we possess no exact estimates in regard to what constitutes an abnormally great or an abnormally small diameter, nor in regard to the influence of age, size, sex, and part of the chest examined in modifying these measurements, it is obvious that numerical calculations made

by callipers would be useless for the purpose of describing the paralytic thorax.

The flattened chest has, on the whole, a drooping appearance; its muscles are thin, and often present a contrast to those of the upper extremities; the intercostal spaces are broad and flat; the suprasternal and infrasternal hollows depressed, and the clavicles very prominent. The manubrium sterni is bent backwards, and forms at the insertion of the second ribs the well-known angulus Ludovici. Since the narrowing affects chiefly the upper parts, the lower portions of the chest appear relatively broader. Posteriorly, one is struck with the projection of the scapulæ, and sometimes with the flat and perhaps concave form of the supra- and infraspinous fossæ. The pathological appearance is still farther heightened by the long, attenuated shape of the neck, and by the depressed and rounded shoulders. The head falls somewhat forward, and the spine sinks so as to be slightly convex backwards.

The chest in phthisis not only presents the characters above mentioned, but in general its defective development contrasts strikingly with that of the rest of the body.

In fact one is often surprised at the small dimensions of the chest in men who appear almost robust when clothed, and also at the poor development of the muscles of the chest as compared with those of other regions. Not infrequently a thick layer of fat conceals this defect, but a careful examination will probably detect it. A woman, who weighed two hundred pounds, and whose brothers and sisters were said to have all been equally colossal, presented herself at the clinic for examination on account of hæmoptysis. She stated to me that she had had a slight cough for only a few weeks, and yet she had already lost forty pounds in weight. Although her external appearance made it improbable that she had consumption, yet on exposing her chest, one could easily see that the configuration was strikingly defective, and the sterno-vertebral diameter small. The elevation movements of the upper parts were very unsatisfactory, but no other signs were discovered on examination, except prolonged expiration over the upper portion of the left lung. She finally met the same fate as her brothers and sisters.

Besides this general deviation from the normal form, a condition which may be regarded both as a cause and as a symptom of apex disease, there are also inequalities in the corresponding portions of the chest on the two sides, more particularly in the upper parts, where the lesions are the most common. Very often the depression, both above and below the clavicle, is more marked, and the anterior upper surface more sunken on one side than on the other. These inequalities are most noticeable when the underlying upper lobes of the lungs have become shrunken, either from the development of cirrhotic tissue, which is the main cause, or from a combination of this condition with cavities.

Inseparably connected with these differences in the configuration of the chest are the *anomalies in movement;* but these cannot always be detected without certain precautions. They may not be noticed in ordinary quiet breathing, but when the patient is made to respire deeply, the excursions of the upper ribs are then seen to be defective, and unequal on the two sides. When the spinal column is normally straight, a diminished and more tardy elevation of one infra-clavicular region indicates a morbid condition of the upper lobe. Sometimes the awkwardness of the patient prevents a satisfactory examination, but this difficulty can generally be overcome. Many patients, when they voluntarily take a long breath, raise their shoulders, and contract other muscles in such a way that it is difficult to form any opinion in regard to the elevation movement of the upper and anterior part of the chest. In such cases the respiratory movements may be increased involuntarily by making the patient walk up and down the room, cough a few times, etc. This imperfect and unequal elevation of the upper chest wall is often seen more clearly when the patient is examined by looking at him from above while he is in a sitting posture. I regard these signs as very important, and I do not recollect a single case, in which the apices were found in a normal condition, or remained so, when the elevation movement was much diminished, or unequal on the two sides. I think it would be desirable, therefore, if we possessed some easy, practicable method, by which the amount of expansive movement might be expressed with numerical exactness, instead of being guessed at. Such a method

would also enable us to form an opinion in regard to the progress of a curative process, and as to whether the gymnastic exercise, or change of climate which had been adopted in the case of young persons with badly developed chests, had been of any real service. Callipers are not adapted to this purpose, while the cyrtometer of Woillez is not suitable for application to the upper part of the thorax, and gives no information in regard to the inspiratory excursions. The spirometer does not indicate whether the upper parts of lungs are receiving more air, and the various apparatus, which have been devised to answer as stethographs, either register inaccurately, or are too complicated for the practising physician.

In consumption the results obtained by *palpation* are unimportant, except in connection with the complications of the disease. This sign is, however, valuable as an aid to inspection. If the patient's clothes fit loosely, palpation will enable us to form a fair estimate in regard to the symmetry and amount of the inspiratory expansion of the upper parts of the chest. For this purpose both hands should be placed upon the shoulders of the patient in such a way that the thumbs lie anteriorly parallel to, and underneath the clavicles, with the other fingers downwards over the scapulæ, while the patient is told to take a full breath.

Over condensations, palpation beneath the clavicle or in the supraspinous fossa often detects an increase of vocal fremitus, which is generally most distinct over cavities. When the anterior border of the left upper lobe is infiltrated, the closure of the semilunar valves of the pulmonary artery can be felt. In complicating pleurisy a rubbing movement may be detected; in pleuritic effusion and in pneumothorax the fremitus is absent, etc.

All these signs obtained by inspection, percussion, and mensuration are in many cases clearly marked even at the outset of the disease; but, as might be expected, they become still more striking in the later stages, when the morbid conditions upon which they depend have increased. These methods of examination are also especially valuable in enabling us to judge whether the case is progressing to a cure.

In health the *percussion note* over the anterior and upper

part of the chest is clear and full. It may also be heard for four or five centimetres above the clavicle on each side of the neck, and in this situation is usually somewhat less loud than it is under the clavicles. In the first, second, and third intercostal spaces towards the axilla the sound is duller, in consequence of the thick muscular layers. It is also somewhat dull in the supraspinous fossa and the inter-scapular regions, but over the scapula the dulness is still greater.

In phthisis the percussion sound will naturally present all shades of dulness, according to the amount of subjacent pulmonary tissue, which is void of air, and in proportion to the nearness of this tissue to the surface. The intervention of pleuritic deposits of much thickness will also have a modifying effect. So also the presence of local emphysema, tense, or contracted tissue, or cavities of sufficient size will produce all the transitions from a non-tympanitic to a tympanitic note. Such a cavity, if it have thin walls, and be situated near the surface, gives a "clapping (klatschende) sound," the bruit de pot fêlé. In pneumothorax the note has a metallic-tympanitic quality.

In a word, the sounds which are developed during the course of phthisis, either in the same patient or in a series of cases, present all possible variations in quality. Percussion also very often excites fibrillar muscular spasms, especially in the pectoral muscles; a sign of common occurrence whenever the nutrition is much impaired.

Corresponding to the lesion, which is most frequently present, the most common alteration in the percussion note is a moderate dulness over the affected side both anteriorly and posteriorly. This dulness is ordinarily noticed on one side only, or is more marked on one side than on the other. Complete dulness, as it occurs in croupous pneumonia of the upper lobe, is very rare in phthisis. Even when the sound is most dull, it is still moderately resonant, and since in most cases there is a gradual formation of cavities, or of emphysematous groups of vesicles surrounding the cicatrized spots, and a continuous contraction of the cicatrices themselves, the note again acquires its clear quality, and in the later stages of the disease not infrequently becomes tympanitic.

In many cases the first change noticed in the percussion note is that it does not reach as high above the clavicle as it should, and when this condition, as verified by auscultation, is found to exist on one side and not on the other, the sign is very significant. The affected apex sinks down so as to occupy less than its normal space, and sometimes the sound elicited over it does not rise more than from two and a half to three centimetres above the clavicle. This diminution in the volume of the apex may however be produced by a shrinking process, which has long since terminated, as well as by recent disease, and consequently this sign taken by itself is undecisive.

The more extensive the parts which are devoid of air, the more marked the dulness. The greatest amount of condensation, however, takes place in cirrhotic degeneration, the chronic pneumonia proper, in which the phthisical process terminates in the development of connective tissue, that is, in the form of consumption which has the most favorable prognosis; hence the most striking alteration in the percussion note over the upper part of the chest does not correspond to the most dangerous amount of disease; but, on the other hand, it might almost be said that a considerable degree of dulness is an auspicious omen.

Besides this lowered position of the apices of the lungs, percussion shows also that in very many cases the first dulness is to be detected in the supraspinous fossa, because the infiltration is usually situated more towards the posterior surface of the lung, while the anterior parts do not become affected until later. During the existence of hæmoptysis, it is advisable either to percuss very gently, or to avoid percussion altogether, as there is nothing special to be gained by it.

As there is no percussion sound which is characteristic of phthisis, so there is no pathognomonic *auscultatory* sign. Now that Laënnec's discovery in 1816[1] and Avenbrugger's percussion, as perfected by Piorry, have been naturalized on German soil

[1] Traité de l'auscult., 4 édit. Paris, 1837, p. 10. The allusion is to his discovery, at this date, of mediate auscultation by means of paper rolled into the form of a cylinder, and applied over the cardiac region of a young woman, in whose case percussion and palpation were of no avail on account of the great degree of fatness.—Tr.

by a Scoda, it is to be supposed that German physicians no longer, like the French, require definite signs for different diseases, and that they are all aware of the fact that the physical signs derived from auscultation can be produced only when the necessary conditions are present, and that, these being present, the former will not fail to be found; in other words, that it is not the disease but the condition of the parts which is disclosed by the physical signs. To utilize these results of percussion and auscultation so as to be able to recognize the special diseases in the organs examined, is a matter of judgment, of skill, of estimation by comparison, and also of knowledge and study.

As was said of the percussion signs, so it may also be said of those of auscultation, that they present every possible variation during the course of phthisis, because all the conditions necessary for the occurrence of the whole range of auscultatory phenomena in the respiratory organs are to be found partly in the bronchial, parenchymatous, and pleuritic lesions, which are the direct result of the disease, and partly in the pneumonia and pleuritic effusion, which occur as complications.

In a disease which, like consumption, usually begins insidiously, and whose lesions almost always consist of numerous small scattered processes; in a disease, moreover, in which our hope of success lies in early treatment, it is, of course, exceedingly important that the first abnormal signs, however inconsiderable, should receive attention; and these signs should be sought for where our pathologico-anatomical experience has shown that the first changes in consumption take place, viz., in the upper lobes and especially in the apices.

In the healthy lung, wherever there is vesicular parenchyma, which is expanded by inspiration and collapses on expiration, the inspiratory sound is heard as a vesicular, prolonged murmur, while the expiratory sound is of shorter duration, and has a gentle blowing quality. Wherever the air is contained within rigid walls, as in the trachea and large bronchi, the breathing is generally bronchial in character both on inspiration and expiration. Bronchial breathing is also normally heard behind the manubrium sterni, and over the upper portions of the interscapular regions, situations occupied not by the pulmonary paren-

chyma, but by the trachea and the two large bronchi. Moreover, even when the apices are healthy, the space which they occupy and their position are subject to variations, because the lungs are not inflexible organs fixed at all points. In these upper portions the position of the rigid bronchial tubes alongside of the pulmonary parenchyma supplies conditions for the production of two different sounds. Can these sounds, when heard simultaneously, lose their twofold character, and coalesce into a third sound; and is it possible, and if so, under what circumstances, for one sound to overpower the other without the presence of morbid changes?

Questions such as these are naturally suggested by the fact that, even in persons with perfectly healthy apices, deviations from the normal respiratory murmur are not infrequently noticed at some distance from the situation occupied solely by the trachea and bronchi.

The *right* supraspinous and supraclavicular fossæ are the special locations in which uncertain or bronchial respiration may occur under normal circumstances, and it is generally acknowledged to be unsafe to draw any conclusion from these signs, when they are heard only in these situations, and there is no other evidence of pulmonary lesion. Every one knows that these anomalies are most frequently met with in thin persons; they occur also in the aged, but this is a fact of less significance in connection with phthisis. When an abnormally loud sound is produced in the upper respiratory organs, it may interfere with the detection of the vesicular murmur, which still takes place in the apices. The beginner should bear in mind, therefore, that the awkwardness of the patient or the existence of disease, *e.g.*, in the larynx, may hinder his forming an opinion as to the condition of the apex. Moreover, it often happens that the sounds in the apices are enfeebled in consequence of the imperfect expansion of these parts by the respiratory movements, as is especially the case in the defective training of the chest previously described.

These sources of error being avoided, the first deviation from the normal character of the respiratory murmur is a prolongation and roughening of the expiratory sound. Fournet and

others have even gone so far as to give the numerical ratios between the lengths of the inspiratory and expiratory sounds, which are indicative of apex disease. Presently the inspiration becomes harsher, and after a while loses its murmuring character; then the expiration acquires a bronchial blowing quality, and finally, when the changes become more marked, this is noticed in both the inspiratory and expiratory sounds. This successive order of changes represents the gradual transition of the parenchyma from an aerated to a non-aerated condition, and phthisis is the disease of all others in which each step of this process can best be traced.

Prolongation of the expiratory sound, and an indistinctness (uncertainty) of the inspiratory murmur are therefore to be regarded as the first changes. To these signs may be added also the difference in the strength of the respiratory murmur as compared with that heard in the immediate neighborhood, or in the corresponding region on the opposite side of the chest. If such inequalities are found to be permanent, and are not removed by coughing and deep inspiration, the sign deserves attention as an early indication of disease.

Another sign of equal significance is the *jerking respiration*, in which a vesicular inspiratory murmur is heard, which is divided into several parts by short abrupt pauses. The sound is in reality an interrupted vesicular respiration, and is very often noticed in persons who afterwards become consumptive; or as the first abnormal change on the healthy side in phthisical patients, who have hitherto presented unmistakable auscultatory signs on one side only; or on the diseased side as an indication of extension of the disease downwards. As to the causation of this sign it is doubtful whether it is a sort of friction sound produced by subpleural nodules, which interfere with the free movement of the pulmonary over the costal pleura, or whether the bronchi are compressed by small scattered tubercles or inflammatory masses of tuberculous (broncho-pneumonic) origin in such a way that at the beginning of the inspiratory expansion the air is unable to force its way through the tubes, but finally succeeds as the expansive movement progresses; these different moments of time, in which the air alternately fails

to enter, and then enters the pulmonary lobules, being recognized by the ear. The existence of this sound is generally acknowledged; its significance of the existence of very minute changes in the pulmonary parenchyma is very probable, and its occurrence in consumptives in situations where unmistakable parenchymatous changes are subsequently found, undeniable.

Of equal and perhaps greater importance are the crackling râles which are heard during the early part of the disease in the supraspinous and supraclavicular fossæ. In many cases these râles cannot be detected in ordinary breathing, but only after the patient has coughed, and they are often so insignificant and so transitory that the unskilled observer often overlooks them. Whether they are due to fluid in the bronchi, or to an alternate adhesion and tearing apart of the bronchial walls, is a question yet to be determined; but the point is one of little practical importance. At the outset of the phthisical process these two conditions—changes in the bronchial walls and fluid within the tubes—are probably always both present. In accordance with the old ideas in regard to the crackling râles, it was formerly held that when a catarrh is limited to the apex it depends upon affections of the parenchyma, and therefore points secondarily to phthisis, and the same significance is attached to these râles to-day. Still another auscultatory sign, from which an abnormal condition of the apex may be inferred, is the existence of a *systolic murmur in the subclavian artery on the affected side.* I have already referred to this point in another place.[1]

The subclavian artery lies transversely in front of the apex of the left lung, where the latter rises above the clavicle, and for about an inch it runs over the anterior surface of this projecting portion so close to the pleural sac that it appears to be attached to the parietal layer. It is very probable, therefore, that when the pleural surfaces in this neighborhood have become adherent to each other the shrinking of the apex may produce a bending or alteration in the direction of the subclavian artery, and that this is especially likely to occur when the respiratory movements are more active than usual. Now, when an elastic tube,

[1] See also *Friedrich's* Herzkrankheiten, and *Gerhardt*, Lehrbuch der Auscultation, 1. Aufl., S. 173.

through which a current is flowing, is narrowed at any point, a murmur is produced; in fact Kiwisch has supposed, though of course incorrectly, that the vascular murmurs, such as the placental murmur and the anæmic murmur (Nonnengeräusch), are to be referred to a compression and narrowing of the different arteries (carotid, epigastric); but however this may be, one can at any time produce a systolic murmur for himself by compressing an arterial branch of some size. Such a systolic murmur is, however, often heard over the clavicle, without there being any disease of the apex. It is very common in anæmic persons, especially in those who are chlorotic, and in such cases it is also noticed that the sound varies with the respiratory movements; that is to say, it increases during inspiration, and becomes more feeble during expiration, or it cannot be heard at all except at the end of a deep inspiration. But when the subclavicular murmur depends upon disease of the apex, the reverse is the case. In cases where the shrinking of the apex could be demonstrated in other ways, I have found that the sound is always loudest during expiration, or only at the end of the expiration. Consequently when a systolic murmur, which is heard in the subclavian region or at the outer half of the supraclavicular fossa, *is detected only during expiration*, or *becomes louder at that time*, I regard it as evidence that the pleural surfaces of the apex of the lung on that side have become adherent.

As the pulmonary disease advances, the bronchial breathing and the bronchophony over the upper lobes become clearer and clearer, and when cavities are present both of these sounds gradually acquire an amphoric echo. If crackling râles are heard, they gradually become sibilant, at first only in single bubbles and then in all. The size of the bubbles increases, and the râles become unequally vesicular and sibilant; but their abundance varies extremely in the same patient at different times. When a true intermission in the phthisical process takes place, the râles become more scanty, and may disappear entirely for some time; but they again increase and often extend further downwards when a renewed attack or an intercurrent inflammation of the bronchial mucous membrane occurs.

Not infrequently the most marked abnormal auscultatory

signs are to be detected underneath the spine of the scapula, or about the middle of the shoulder-blade. This is to be explained by the fact that very often the morbid process is more advanced in the upper part of the lower lobe than in the lower portion of the upper lobe. In many persons the apex of the lower lobe occupies a very high position, as high as the spine of the scapula, especially on the left side.

When the cavities have become larger, and especially when they are situated superficially, we not infrequently hear also what has been described by Seitz[1] as "metamorphic respiration" (metamorphosirende). I had always supposed that this sound was generally known, because I had been familiar with it since my course of instruction in auscultation under Traube in 1845, and had very often demonstrated it to my pupils, always stating to them that it was Laënnec's "souffle voilé."[2] And yet I see that Laënnec's description of the latter, and of the conditions under which it is heard, do not quite conform to the character of this sound. At all events it is very characteristic, and must certainly be regarded as one of the most useful signs of a cavity.

If the cavity be of very large size, extending from the apex of the lung almost to the base, by the coalescence of several cavities, metallic tinkling may also be heard. This sound is generally heard only in pneumothorax, because it is necessary for its production that the cavity should be very large and should have solid walls—conditions which very seldom occur except in this affection.

Moreover, the movements of the heart may produce murmurs in a cavity situated near that organ, or the cardiac sounds may acquire, under these circumstances, a strikingly sibilant and reverberating character. Probably, also, vascular murmurs of a different origin may arise in such cavities, and become audible in consequence of the reverberation.[3]

Sometimes it is possible to ascertain the shape of the cavity. If the longest diameter of the latter be from above downwards,

[1] Deutsches Arch. f. kl. Med., I., S. 292.

[2] L. c. Vol. I. p. 76.

[3] *Gerhardt*, Lehrbuch der Auscultation, S. 173. *Bartels*, Deutsches Arch., Bd. VI.

the percussion sound changes according to the position of the patient, whether he is sitting or lying down (Gerhardt).

The subject of *spirometry* has already been alluded to, and the description which has been given of this method of diagnosis shows that it may be made available in the beginning of the disease. If a person who has been coughing for some time, and is suspected to have phthisis, but presents none of the above signs of apex disease, be found not to have the normal minimum of vital respiratory capacity which belongs to his weight, size, and age, but rather to fall about 500 C.C. below the same on repeated experiments, this fact increases our suspicion; on the other hand, our fears will be allayed if he attain more than this minimum.

Quite recently Waldenburg[1] has employed pneumatometry in cases of phthisis, and has found, as might have been expected, that both the inspiratory and the expiratory pressure are below the normal. The insufficiency of the latter is, however, much less than that of the former; this relation being the reverse of that which is found in emphysema, where the insufficiency of the expiratory pressure is greater than that of the inspiratory. Waldenburg hopes that this fact will be a valuable aid in distinguishing phthisis from bronchial catarrh.

Laryngeal Symptoms.

The functional derangements of the larynx in phthisis will be considered here only in a general way, because the subject of the first appearance and further development of tuberculous disease in these parts, as studied especially by direct inspection, has been presented in detail elsewhere.

Aside from the cough, which may, of course, be caused partly, and in some cases chiefly, by the laryngeal affection, when the latter exists as a complication of phthisis, the only other local symptoms to be considered are the alterations in the voice, the signs of laryngeal stenosis, and the imperfect closure of the larynx during the act of swallowing.

Hoarseness of the voice and cough are very frequent symp-

[1] Berliner klin. Wochenschr., 1861, S. 541.

toms. As it is often noticed very early, indeed before any chest symptoms occur, and becomes more and more prominent and severe as the disease advances, it attracts the attention of the patient and of those about him. Very naturally, therefore, laryngeal consumption is regarded by the public as a special form of the disease. This view has always been held by physicians also, and is still held at the present time; that is, so far as believing that the first symptoms of phthisis sometimes make their appearance in the larynx. This question, however, is very much the same as the similar one in regard to hæmoptysis; it is impossible to get at the truth, because we are entirely unable to fix the exact time when the pulmonary disease begins, and the latter may exist a long time without any symptoms.

There is no doubt, however, as to the incorrectness of the French view, that the laryngeal affection is produced originally by infection from substances coming from the lungs. On the contrary, it is more probable that infecting material is carried downwards from ulcerating surfaces in the larynx.

Hoarseness does not, however, always depend upon tuberculous disease of the mucous membrane; it may result also from acute or chronic catarrh without ulcerations, a cause which may be only temporary. Not infrequently *syphilitic* ulcerations occur in consumptives, or there may be mixed forms of ulceration, the exact nature of which it is very difficult to recognize.

That laryngeal and pulmonary syphilis may run its course under the form of phthisis, is unquestionable. The infiltration of the apices may be of a syphilitic nature, and the affection may hasten to a rapidly fatal termination with hectic fever, or at least the patient may be brought to a condition of great emaciation. In such cases the syphilitic nature of the affection is first discovered by the examination of the throat and larynx, and the suspected phthisis may probably be cured by the appropriate mercurial treatment.

I have reported elsewhere a very striking case of this kind. The patient was a woman about thirty-five years of age, who was admitted to the Greifswald clinic extremely emaciated, completely aphonic, and suffering from hectic fever, with profuse perspirations. The large cicatrices found in the throat made a

laryngoscopical examination necessary; this revealed considerable loss of substance in the epiglottis, and deep, sharply defined ulcerations on the vocal cords, lesions which we were obliged to regard as evidently syphilitic. Besides this, there was also an extensive infiltration of the right upper lobe. Although the woman seemed to be *in extremis*, I used inunction, and not only did the larynx heal, leaving behind thick, radiating cicatrices, but the infiltration also diminished in size, the fever disappeared, the nutrition improved, and for several years afterwards the woman was exhibited to the class in laryngoscopy; her subsequent fate is unknown to me. Last year I saw a very similar condition of things in a girl thirteen years of age, but here iodide of potassium was sufficient to cure the laryngeal and pulmonary affection. A colleague in Wesel informed me that he has cured or relieved by anti-syphilitic treatment quite a number of cases of supposed consumption in syphilitic families.

The larynx is the locality *par excellence*, in which syphilis and phthisis intermingle and intersect each other, and this subject might properly be considered here, but our material seems to me still insufficient for a thorough discussion of the mutual influences which they exert, or to enable us to distinguish syphilitic phthisis from the ordinary form.

The hoarseness, which results merely from catarrhal disease, or from syphilitic ulcerations, may disappear after a time; but this result is very uncommon, where, as is usually the case, this symptom depends upon tuberculous disease or ulcerations of the same character. The yellowish nodules may be seen to break down into small ulcers which spread especially superficially, and sometimes cover the whole internal circumference of the larynx. Sometimes, to be sure, even in these cases the hyperæmia and the tumefaction of the edges diminish, the spreading process is arrested, and the ulcers begin to heal, but a complete cure is very rare, and one of long duration still rarer. Consequently the hoarseness, which depends upon tuberculous processes, steadily increases, although at various times a certain amount of improvement may be noticed, and finally complete aphonia occurs, which lasts to the end of life.

Besides its dependence upon the gradual extension of the

affection of the mucous membrane, the degree of hoarseness will naturally vary according to the actual situation of the lesion. The earlier the vocal cords themselves, and especially their insertions into the processus vocalis, become affected, the more rapidly will the alterations in the voice be produced, even when the lesion is comparatively slight.

Moreover, a certain force in the expiratory current is necessary for the production of a clear vocal sound; hence the amount of aphonia will bear a direct relation to the degree of weakness in the respiratory muscles, and hence, also, in very feeble persons the aphonia may be quite marked, although the local lesions are unimportant.

Besides the hoarseness, consumptives often suffer from *difficulty in swallowing*. Every act of deglutition excites pain. The cases in which the pain depends upon *pharyngeal affections* of a tuberculous character, are, however, quite uncommon. Still such instances do occur, and I have several times seen ulcerations of this nature extend forwards into the cavity of the mouth. I have also had the opportunity of observing, in Greifswald, the further progress of Niemeyer's case of tuberculous destruction of the tongue, and I have even seen these affections of the mouth and throat without any laryngeal disease; but these cases are exceptional.

The *difficulty in swallowing* generally proceeds from disease of the larynx. The occurrence of this symptom is determined entirely by the situation of the laryngeal lesion. As soon as the tissues covering the arytenoid cartilages and the bridge between them become very hyperæmic and swollen, or are removed by ulceration, pain is excited by the passage of food over the parts, and by the pressure of the pharyngeal constrictors. Sometimes this pain is so severe that the patient is afraid to eat, and rejects all nourishment. Unfortunately this distressing condition occurs in many cases at a very early period, and then inanition advances rapidly; in other cases, however, these symptoms do not set in until towards the close of life. The dysphagia, which makes its appearance late in the disease, usually depends, not upon the deeper-seated tuberculous affections, but upon round erosions, which, as Louis has explained,

seem to be produced by erosive inflammation in these parts resulting from the almost continuous adhesion of masses of pus coming from profusely suppurating cavities.

In many patients the act of deglutition is not only painful, but it may also be actually prevented by the cough excited, especially when they attempt to swallow liquids. This happens only when there is deep destruction of the vocal cords, making their perfect apposition during deglutition impossible. Traube's[1] experiments have shown that it is not the office of the epiglottis to effect a complete closure between the digestive and the respiratory apparatus during deglutition, and that, on the contrary, the latter may be satisfactorily performed even in the absence of a considerable part of the epiglottis. In all cases, therefore, where the inability to swallow is not merely temporary, and dependent upon some accidental disturbance in the mechanism of deglutition, but is of constant recurrence, we may infer that the difficulty proceeds from defective closure of the vocal cords.

Finally, laryngeal diseases, as they occur in consumptives, may also produce symptoms of *laryngeal stenosis*, such as stridor and dyspnœa. The causes of this condition are various; sometimes they are temporary, at others permanent. In the former case they consist of inflammatory and œdematous swellings in the neighborhood of ulcers, which give rise to symptoms of œdema glottidis, the inspiration being interfered with more than the expiration; but, as the mirror shows, these symptoms generally continue for several days before they become threatening. The permanent narrowing of the lumen of the larynx may be produced, among other causes, by a rigid condition of the vocal cords, which are also generally swollen, and which thus act as a septum to divide the cavity of the larynx into an upper and a lower half with only a narrow opening between them posteriorly. The stenosis may also be due to various other conditions, such as considerable chronic thickening of the edges of ulcers, polypoid proliferations of the same, dislocated fragments of the cartilages, etc. This narrowing of the larynx, whether from temporary or from permanent causes, must, of course, hasten the fatal result.

[1] Beiträge zur experiment. Pathologie, Heft I.

That all three of these functional disturbances, hoarseness, dysphagia, and dyspnœa may occur together, is obvious without further explanation. In most cases aphonia is the only one of the symptoms present, but the other two are never met with without preceding aphonia. There is, however, no causal connection between them, and the aphonia may be associated with either dysphagia or stenosis alone.

Symptoms of the Organs of Circulation.

In phthisis the contractions of the heart are very generally increased in frequency. Even before the onset of the disease the pulse is found to be quickened, especially in young persons, and this without any corresponding elevation of temperature. In many cases this symptom may be explained by the marked preceding anæmia, since it is well known that all anæmia tends to increase the contractions of the heart. The acceleration of the pulse is frequently more noticeable in the upright than in the horizontal position. A slight amount of exercise or of mental excitement produces an undue frequency of the pulse, increased force of the cardiac impulse, and palpitation, which is not only felt by the patient but may be noticed by others; symptoms which are all met with in young persons as a result of every form of anæmia.

The pulse is not only rapid, but it is also always soft and empty, and even when fever is present we fail to find the tension which is present in ordinary inflammatory fever.

When the contractions of the heart are quiet and the frequency of the pulse is normal, even during the evening, it may be inferred either that the disease is making but very slow progress, or that an arrest of the process has already taken place.

During the course of phthisis, in consequence of the breadth and thinness of the intercostal spaces, there is often to be seen a sinking inwards in the cardiac region at the time of the systole. It is only necessary to be aware of this sign to avoid confounding it with the similar appearance which is noticed only at the situation of the apex beat in cases where the pericardial surfaces have become adherent. This latter appearance results from the

dragging inwards effected by the adherent apex as it performs its systolic downward movement, while the systolic sinking alluded to, which occurs at other parts of the cardiac region, is due to the atmospheric pressure, to which the broad and thin intercostal spaces yield when the action of the heart is sufficiently forcible.

When consolidation of the left upper lobe is followed by shrinking, the impulse of the heart is felt over a broader space and is often displaced upwards and to the left. So also a pulsation may be seen and felt in the neighborhood of the pulmonary artery, and the sharp closure of the semilunar valves may be appreciated by the hand as a diastolic shock in certain cases where the anterior and upper extremity of the upper lobe is infiltrated and transmits to the chest wall the sounds and movements of this vessel. Sometimes, also, a systolic murmur may be generated in this neighborhood, which seems to be due to compression. The diastolic pulmonary sound often has a clear, sibilant quality, although there are rarely any signs of hypertrophy of the right ventricle; but, on the contrary, the heart at the autopsy is found to be diminished in size, not only from the loss of a considerable amount of fat, but also from the imperfect filling of its cavities and the wasting of its muscular substance.

In consequence of the diminished fulness and tension of the arteries throughout the body, audible systolic murmurs may be excited by slight causes in any part, and there is probably no doubt that even the occurrence of such sounds in the chest, especially over the upper lobes, as reported by different observers, particularly Bartels,[1] is to be explained by compression. At all events, the existence of infiltrated masses in the lungs, of enlarged bronchial glands, of circumscribed mediastinal pleurisies, etc., furnishes abundant opportunities for the occurrence of such a local narrowing of a branch of the pulmonary artery.

In children, in whom the affection of the bronchial glands is frequently the chief lesion (Rilliet et Barthez),[2] a considerable

[1] Deutsches Arch., Bd. VI.

[2] Traité clinique et pratique des maladies des enfants. Paris, 1843, T. III., p. 164 et seq.

amount of compression may occur. In Greifswald I saw a child whose aorta was compressed in this way to such an extent that the left ventricle had become hypertrophied.

The venous branches within the chest are still more readily compressed, and this is the most frequent explanation of those cases in which dilatation of the veins on the neck, shoulder, and chest is noticed. This condition is more distinctly seen in young consumptives, with a thin and generally pale skin. To the same cause may be attributed also the cyanotic color, the swelling of the face, and many asthmatic attacks.

In this connection the signs of anæmia may be considered. This condition, which is often one of the first indications of the approach of phthisis, develops with various degrees of rapidity during the progress of the disease, and often reaches a marked degree of intensity, although the patient takes a sufficient amount of food and his excretions do not appear to be excessive. In tuberculosis, therefore, as in carcinoma, the anæmia has been regarded as due to the unexplained influence of a malignant neoplasm. Ultimately hydræmia is developed, and, under the co-operating influence of obstruction to the return of the venous blood, œdema occurs, which is first noticed about the ankles, disappearing in the morning after elevation of the lower extremities during the night, and then gradually increasing to more formidable dimensions.

Here also may be mentioned the arterial emboli, which originate in coagula within the pulmonary veins. Embolism from this source has been repeatedly observed, even in the brain, when cavities have existed for a long time.

In former times it was supposed that in the majority of cases of phthisis the *lymphatic system* shared largely in the morbid process, and this view was confirmed by the pathological anatomy of the disease; but aside from the signs of pressure exerted by the bronchial, mesenteric, and retroperitoneal glands upon the neighboring organs, the signs of these affections are generally very obscure.

In some cases when palpation shows that the lymphatic glands in the clavicular region or in the axilla are enlarged, or tumors are discovered in the abdomen, which we are obliged to

regard as of the same nature, we may infer with some degree of probability that the glandular affection has extended to other parts inaccessible to direct examination, and that the dulness, which is detected over the manubrium sterni, or at its sides, or between the clavicles, may be due to this cause. But at present we are unable to show either what influence these glandular enlargements exert upon the course of the disease by obstructing the current of lymph and chyle, or even what symptoms are produced by them.

Since the term pulmonary consumption is used only to designate the most conspicuous and most frequent localization of a constitutional disease, the affections of other organs, in which the same morbid process is of frequent occurrence, should be regarded as symptoms rather than as complications. On the other hand, to discuss in detail the disease as it occurs in each organ would necessarily involve much repetition, and, accordingly, in the following remarks upon the digestive organs, I shall confine myself to points of essential importance.

In the *stomach* genuine tuberculous disease is so rare, and, when it does occur, so limited in extent, that it never produces recognizable symptoms. In a considerable number of cases nothing abnormal is found at the autopsy, but in many others lesions of the mucous membrane are present, consisting of anæmia, hyperæmia, softening, or either acute or chronic catarrh. The gastric functions correspond to these conditions: sometimes there are no symptoms of disturbance; in other cases there are either temporary derangements, or chronic difficulties of varying degrees of severity.

Besides the various symptoms of gastric catarrh, which it is unnecessary to describe here, as they present no characteristic peculiarities, the *anorexia*, which is noticed at the outset of the disease, and sometimes continues during its whole course, deserves special attention. This symptom is present more particularly in the acute forms and stages of the disease, and is commonly ascribed to the accompanying fever. This explanation (*sit venia verbo*) is, however, unsatisfactory, because in consumption, even during its febrile stages, the appetite is very often undisturbed.

Whether this diminution in the amount of nourishment taken may not, in some cases, be rather a cause than an accompaniment and result of the constitutional disease, is a question difficult to determine. There is probably no doubt that inanition from any cause tends to the development of consumption, and that a person predisposed to this disease will become affected more speedily and with more certainty when he lives upon an impoverished diet; but, on the other hand, the cases are not uncommon in which anorexia continues for a long time without any further symptoms being noticed. Other patients suffer also from *nausea*, occasional *vomiting*, and *pain* or oppression in the epigastrium; symptoms which, taken together, indicate the existence of catarrhal inflammation. At the same time the tongue is generally coated and the epigastrium sensitive to pressure.

Vomiting and *pain* in the epigastrium may be excited by severe paroxysms of coughing. The pain which is thus produced is to be regarded as a myalgia, while the vomiting which ensues upon coughing is probably due to a variety of causes.

The nervous centre, from whose irritation the reflex act of coughing proceeds, is situated very near the centre concerned in the act of emesis; the irritation probably passes readily from the one to the other, and the more readily when the existence of pharyngeal and gastric catarrh keeps the vomiting centre in an irritable condition. This statement accords with our daily experience.

Now one of the conditions essential to the production of vomiting and evacuation of the contents of the stomach is that the cardia shall be open, and this may probably occur just as well from a paralytic as from a spastic state of the part. The fact that in health the dilatation is spastic is no evidence that it is so in disease. The numerous cases, in which air or even fluids in the stomach regurgitate at any moment into the œsophagus, without there being any evidence of tension of the walls of the stomach, make it very probable that vomiting may in some instances be due to an incomplete closure, a paralytic condition of the cardia. This condition being present, it is easy to see that prolonged coughing, which certainly produces considerable

compression upon the abdominal viscera, may also force the contents of the stomach upwards.

Anorexia, aversion to all food of a nourishing character, and vomiting are, at all events, very serious symptoms, and their relief by treatment is a point of essential importance. Not infrequently the beginning of the disease is masked by such gastric derangements, and the phthisis is regarded as merely a dyspepsia with anæmia, or as a chronic gastric catarrh.

In many cases there are no gastric disturbances of any kind from the beginning to the end, and up to the last moment the good appetite and painless digestion seem to give some foundation to the hopes of the patient. This is more frequent in the protracted cases than in the acute. Such instances certainly show that the wasting in phthisis is not solely dependent upon the amount of food taken.

But even when the anorexia, pain, and vomiting have continued to the end of life, the autopsy does not always reveal adequate lesions of the gastric mucous membrane; and, on the other hand, when the appetite and digestion have been unimpaired, the membrane may be found to be diseased; but it must be admitted that in recent times the condition of the stomach in phthisis particularly has not received much attention. Its more *minute* examination seems to be attended with very great difficulties, and very likely the stomach is one of those organs in which even serious functional derangements may occur, independently of permanent structural changes which can be demonstrated post mortem. In conclusion, it should be mentioned that towards the end of life a distressing aphthous condition of the cavity of the mouth and throat may be developed, especially in patients who have suffered much from gastric derangements. Such a condition always points to a speedy termination of the disease. In other respects the state of the buccal cavity and the coating of the tongue are only such as are usually symptomatic of the digestive derangements and the constitutional condition, and need not be described here.

Thirst is a very inconstant symptom. Sometimes it is obviously connected with gastric disturbances, fever, and perspiration; at other times with diarrhœa, but it may occur indepen-

dently of these affections, or may be absent, notwithstanding their presence. It is never a very marked symptom, nor does it occasion much distress.

On the part of the *intestinal canal* the symptoms consist of *pain* and *diarrhœa*.

The *pain*, whose situation corresponds to the position of the small intestine, unquestionably proceeds in many cases from actual disease of the intestinal wall. In many cases it does not occur spontaneously, but only on palpation, and then especially in the cæcal region and hypogastrium; localities which generally correspond to the situation of the intestinal ulcers. The infrequency of any considerable amount of intestinal pain in phthisis may be explained by the fact that the mucous membrane is not particularly sensitive, especially when the lesions have formed gradually and have continued for some time, and that when pain does occur, it is probably due rather to severe muscular contractions and irritations of the serous peritoneal coat. These pains may be either migratory or fixed; in the latter case they correspond to the situation of the lesions, which generally begin in the lower part of the ileum, where they are most marked, and then gradually extend upwards towards the jejunum, or even within it.

When the ulcerative process takes place in the large intestine it advances in the opposite direction, from the cæcum towards the sigmoid flexure, and is not infrequently attended by wandering colicky pains. But since in the majority of cases in which tuberculous ulcerations of the intestine are found, the peritoneal coat has already become implicated in more or less numerous places either in the inflammatory or in the tuberculous process, it is impossible to determine whether the pain is due to the lesion of the mucous membrane or to that of the serous investment. That it is generally due to the latter cause is evident from the fact that the early stages, and frequently even the advanced condition of ulceration of the mucous membrane, are met with in very many persons who have never experienced any abdominal pain. In some cases the abdominal pain clearly depends upon other lesions than intestinal ulceration; as, for instance, acute peritonitis from perforation, or the chronic from

of this affection. These will be considered among the complications.

Far more frequent than this symptom is the occurrence of *diarrhœa*. In one hundred and twelve patients, Louis noticed its absence in only five. Although this proportion is larger than is generally the case, there is no doubt that the symptom is very frequent. It may arise independently of any ulcerative process in the intestinal mucous membrane, just as in the respiratory organs expectoration occurs, which is unconnected with the tuberculous process; and even when ulcerations are present the diarrhœa may depend upon the intestinal catarrh, which is always associated with them.

As a rule, however, the number and the quantity of the discharges are in direct proportion to the extent of the intestinal consumption. The more exclusively the process is confined to the small intestine, the less abnormal in most respects are the discharges; on the other hand, the greater the implication of the colon, the more fluid and frequent do they become. In some cases during the whole course of the disease there are not more than two or three evacuations of a pulpy consistence daily, while in others there are twelve or more watery discharges, which may or may not be painless. On the other hand, post-mortem lesions of considerable extent are sometimes found in the intestine, without there having been any diarrhœa during life. Solid, globular, normally colored fæces may often be found in these cases within the cæcum adherent to the ulcerating surface.

The *character* of the evacuations gives us very little information. Doubtless something might be learned from their examination, if one had the necessary self-denial and perseverance for such an undertaking, but the importance of the subject scarcely warrants the trouble. Pus and blood may be found in variable quantities, coming from the surfaces and edges of the ulcers, and in many cases the pus is sufficiently abundant to be seen as bright yellow streaks running along the bottom of the vessel, when the latter is carefully inclined to one side. Sometimes, when the blood comes from ulcers situated high up in the intestine, the evacuations are of a dirty reddish-brown color, the cause of which can be ascertained only by the discovery of

blood-corpuscles on microscopical examination; but when the blood proceeds from ulcers in the lower bowel, and has not had time to be mixed with the contents of the intestine, it appears in the form of small clots or streaks.

The *odor* of the diarrhœal discharges in phthisis is very offensive when there have been long-continued ulcerations; but where the latter are of recent date, or of slight extent, there is no special change in this respect.

The *color* varies; very often it is a light yellow, with a shade of gray, indicating a deficiency of bile and probably a fatty liver. This color may, however, depend upon the nature of the food, especially when the diet is regulated with reference to the existing diarrhœa. If the ulcerative process be extensive, and the intestinal affection continue for a long time, the bowels become more voluminous, and the middle and lower abdominal regions swollen, doughy, and sensitive to pressure.

Diarrhœa sometimes precedes the other symptoms of phthisis. But in these cases, just as with the laryngeal symptoms and the hemorrhage, it is impossible to decide whether the diarrhœa is merely an early symptom of the disease, or the cause of the pulmonary affection, which is recognized later. The instances in which persons, after suffering for months or years from an uncontrollable or constantly recurring diarrhœa, without any indication of other disease, especially in the respiratory organs, finally succumb to a consumption of the lungs, are certainly rare, but I have myself repeatedly seen such cases. Here the intestinal disease is the main affection, and has the longer duration.

In other instances the intestinal symptoms do not appear until after the respiratory affection has manifested itself unmistakably for some time. If these symptoms are of only moderate severity, and the diarrhœa is not very profuse, the bowel disturbance may amend, or disappear, even after it has continued for months, and the autopsy will now and then show the existence of reparative processes in the intestinal ulcers.

Very commonly the disturbance of the bowels belongs to a later period. In this case it usually sets in with greater violence, rapidly exhausts the patient, and hastens the fatal col-

lapse. During its course, however, the pulmonary symptoms not infrequently abate, the cough and oppression in breathing almost disappear, and although the end is rapidly approaching, the patient thinks that he is much better. Sometimes the tuberculous ulceration terminates in perforation, followed by a general and usually rapidly fatal peritonitis; and this accident is often the first intimation of the existence of intestinal ulcers.

At other times there is an entire absence of diarrhœa, the bowels are sluggish, and the severe straining at stool brings away scybala streaked with a little blood, or produces slight swelling of the veins around the anus, so that at the outset of phthisis and even in its more advanced stages there may, perhaps, seem to be some reason for regarding the case as one of hemorrhoidal disease.

No one who has mingled much with his medical brethren of different generations, will think it superfluous for me to sound a warning here against resorting to that refuge of ignorance, the hemorrhoidal affections, for an explanation of cerebral, cardiac, or pulmonary symptoms, and against regarding the insignificant difficulties so common about the anus as hemorrhoids, and thus confirming the patient himself in what may perhaps be a dangerous error. Such a diagnosis involves the employment of remedies to develop the hemorrhoids, and bring on a flux, and accordingly purgatives, leeches, and violent exercise are recommended; any cough that may be present is regarded as a subordinate matter, until finally these efforts are perhaps brought to an end by the occurrence of pneumonia or pleurisy, and the pulmonary affection rapidly develops.

That genuine hemorrhoids and the hemorrhoidal flux have some influence in the production of bleeding from the lungs, I have admitted in my remarks on hæmoptysis, but these affections have no causal connection with pulmonary consumption itself.

The relations in *fistula in ano* seem to be somewhat different. This lesion is quite common in consumptives, and there is a traditional belief among practising physicians that it has a protecting influence against phthisis. So long as the patient has a suppurating rectal fistula, it is held, he will either remain

free from consumption, or at least will resist its advance, and therefore such a fistula ought not to be closed in a person suspected to have phthisis, still less when symptoms of pulmonary disease are already present.

There is no statistical evidence in favor of this belief, and hence there will continue to be a difference of opinion; but the opinions of our forefathers on this subject are not to be scouted, for certainly it is unfair to deny that they possessed a fine talent for observation, even if they were ignorant of the application of auscultation and percussion to the diagnosis of chest diseases.

I shall refer to this point again under the head of therapeutics.

In the course of phthisis there are two varieties of *liver disease*, the *fatty liver* and the *amyloid liver*. Both occur during the progress of various affections, always develop slowly, and in the more marked cases produce an enlargement of the organ, sometimes to a considerable size. These affections are painless, and as neither the blood-vessels nor the bile ducts are compressed, there is an absence of ascites and icterus; but the secretion of bile is diminished, and this accounts for the lighter color of the contents of the bowel. The anæmia and loss of strength are undoubtedly aggravated by the intercurrence of the liver affection; but as these symptoms are primarily connected directly with the phthisical affection, they cannot be regarded as indicative of the hepatic lesion. The enlargement of the organ is the only diagnostic sign.

In the fatty disease the consistence of the liver is diminished, and, as a rule, its longitudinal diameter is, to a certain extent, increased at the expense of its thickness. At the same time its edge, at least that of the right lobe, is more rounded. The diagnosis by palpation and percussion rests upon these peculiarities. In the case of fatty liver palpation is more difficult; but if the edge can be felt projecting several centimetres below the borders of the ribs on the right side, it will be found to be no longer sharp but blunt. Percussion sometimes brings out the tympanitic sound of the underlying colon. The right hypochondrium is not distended. Besides this, the spleen is unaf-

fected, and there is no increased dulness over the left hypochondrium.

In amyloid disease the conditions are quite different. The organ is enlarged in all dimensions, the edge is sharp, the left lobe is always affected equally with the right, while the spleen is usually at the same time degenerated and hypertrophied. Consequently in amyloid disease the right hypochondrium is more distended and resistent; the sharp edge is situated lower down, and can very readily be felt, while the smooth insensitive organ is more resistent to pressure, and can easily be traced into the left hypochondrium. The percussion note over the liver is everywhere very dull, and the spleen presents various degrees of enlargement.

This form of hepatic disease is, however, not so much a symptom as a complication of a general character, involving other organs also, and exerting a decided influence upon the course of the phthisis; it will therefore be considered again when we come to speak of the complications. As regards the fatty disease in consumptives, this is probably due partly to the fatty food which the patient uses, and partly to absorption of the fat of the body, because the enlarged condition of the organ compels us to suppose that fat is deposited in the cells; that is to say, it is derived from external sources.

The nature of the amyloid affections is still more obscure.

The only diseases of the *spleen* which are found in phthisis are the amyloid degeneration, already mentioned, and acute tuberculosis, which is also quite common, and produces hyperæmic enlargement of the organ.

The *urinary organs* may be diseased in two ways; either primarily, in the form of intense tuberculous inflammation (tuberculosis of the kidneys and urinary organs); this variety will be considered in the ninth volume; or the kidneys may take part in the general amyloid degeneration.

In tuberculosis of the urinary apparatus there is always considerable pain, which gradually extends from the bladder (the prostate and seminal vesicles) along the ureter, on one or both sides, towards the region of the kidneys. The urine usually contains large quantities of mucus, pus, detritus, and blood,

with a certain amount of albumen, and is turbid at the moment of discharge. There is tenderness on pressure over the bladder, perineum, and lateral regions of the abdomen near the kidneys. Micturition is painful, and frequently very urgent.

In the amyloid disease these symptoms are all absent; the urine contains a large amount of albumen, but otherwise its physical properties usually deviate but little from the normal.[1] It is of an intermediate yellow color, and slightly turbid, or clear. The specific gravity is diminished, the quantity about normal, and there are no characteristic organic ingredients.

As soon as the kidneys become affected by the amyloid disease, the paleness of the integuments increases, the patient acquires a waxy pallor, and, as Meckel[2] has observed, the pulmonary symptoms, however marked they may have been up to this time, now recede; in fact the morbid process itself undergoes a regressive change; "the tuberculosis is suddenly changed into the amyloid disease."

In other cases the urine presents only the characters which are usually given to it by fever, gastric derangements, or anæmia.

Nervous Symptoms.

Unless there is a development of tubercles within the cranial cavity, in the form of either diffused tuberculosis of the meninges or of tuberculous masses in the cerebrum, the only intracranial lesion found at the autopsy is anæmia. The longitudinal sinus is empty, or only partially filled; the meninges are pale, with perhaps a little serum between the convolutions and upon the upper surface; the brain substance is more or less softened from serous infiltration, and anæmic. The serous effusion takes place always during the last few days of life. The unimportance of these lesions has consequently led, especially in large hospitals, to an habitual neglect to examine the cranial cavity. As might be expected from the negative character of these lesions, the cerebral functions are fairly well maintained during life.

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[1] *Traube*, Gesammelte Abhandl., II., 378.

[2] Die Speck- oder Cholestearinkrankheit. Annalen d. Charité, IV. Jahrg.)

The mental powers share to a certain extent in the general exhaustion of the body, but rather quantitatively than qualitatively. In fact the individual peculiarities seem to increase with the physical prostration. Persons of gentle disposition become more amiable, and the rude more rude. Almost universally the patient misjudges the nature and progress of his disease. A colleague of long experience made the striking remark to me recently, that he had met with few consumptives who, within a few weeks of their death, had not ordered new shirts to be made. Even physicians themselves, when they are consumptives, are subject to the same peculiarities; and it has always seemed to me that the special attention which many pathological anatomists devote to the disease, of which they are the certain victims, is to be explained in the same way (Meckel, Reinhardt).

The somnolence and slight disturbances of consciousness, which sometimes occur during the last few days of life, are due to the œdema.

Whenever phthisical patients present decided symptoms of cerebral disturbance, it is safe to infer an extension of the tuberculous process to the brain. The occurrence, therefore, of a fixed headache, sudden delirium, or maniacal excitement, may be regarded as very ominous symptoms. The detailed consideration of the individual brain symptoms which arise in these cases of meningeal and cerebral tuberculosis belongs elsewhere.

Affections of the *spinal cord* and *peripheral nerves* are very uncommon. When present they depend upon some incidental local disease. The former, as in the cranium, may be produced by meningeal tuberculosis, or by tuberculous masses in the vertebral canal; the latter by compression and dragging of the filaments of the peripheral nerves by the lymphatic glands, pleuritic deposits, etc.

Cutaneous Symptoms.

The transparency and paleness of the skin, and the distinctness of the veins, which are often noticeable in the prodromal

stage, become more and more conspicuous as the anæmia increases in the later periods of the disease. Such patients are sensitive to slight changes of temperature, and very subject to cutaneous vaso-motor paralyses ; their faces flush on the slightest excitement, and they perspire profusely with even moderate fever. Whether the sweating of phthisis is always preceded by an elevation of the temperature is still undetermined. The fact that this symptom occurs in all convalescents from acute diseases, and in many other persons enfeebled from other causes, and that it is especially common during sleep, is opposed to this view. In consumptives, sweating occurs after every short nap in the daytime, and in no other affection is it so constant and profuse. Meanwhile, in the absence of more definite knowledge, we can only say that this functional derangement of the skin seems to be a peculiarity of phthisis. Of course the great influence of fever in causing and increasing the perspiration is not to be ignored.

During the course of the disease this symptom at times remits or disappears entirely, and so long as the general nutrition is well maintained it assumes but little prominence ; but towards the end it generally increases again, and in the acute continuous cases it appears in its fullest development.

Sometimes the perspiration is accompanied by eruptions in the form of miliary vesicles, or sudamina, but these are uncommon. It is not improbable that the night-sweats are often occasioned or increased by the severe coughing, which occurs unnoticed by the patient during sleep, but attracts the attention of those about him. The perspirations may also be increased by the diaphoretic action of the morphine which is used to quiet cough.

Whether the perspiration in phthisis has any special qualities is unknown. Patients very commonly blame it as the cause of their peculiar lassitude.

The precise relation between the sweating and the pityriasis versicolor, which occurs in scales upon the chest of consumptives, is as yet undetermined. In some cases the conditions resulting from the perspiration, the smearing the chest with fatty substances, and the infrequent change of the undergarments are

obviously favorable to the production of the parasite; but the eruption is also noticed in the absence of all these causes.

The affix "tabescentium" is not always appropriate, because the pityriasis is often noticed very early in the disease, before any emaciation has taken place. These vegetations rarely cause any other annoyance than at most a slight itching. They usually spread in various directions as the phthisis advances.

In the chronic cases, except when there is spinal disease, *bed-sores* are comparatively uncommon, and with proper care may be avoided. They do not belong to the symptomatology of the disease.

The *œdema* of the subcutaneous connective tissue, which occurs in the later stages of the disease, is produced either by an obstruction of the circulation, or by thinness of the blood serum, or by both causes combined. That the obstruction is rarely situated centrally, that is, in the pulmonary circulation or in other organs, is evident from the absence of urgent dyspnœa, or of signs indicative of venous congestion elsewhere. The œdema seems to depend rather upon a diminished *vis a tergo*, which induces a retardation of the venous current in distant parts, and may perhaps lead ultimately to the formation of coagula in the larger veins, the crural and saphenous. Local obstructions may also occur from pressure. Glandular tumors, or the prolonged maintenance of one position, may produce compression of the veins in one of the upper or lower extremities. The thinness of the blood is an accompaniment of the anæmia, and becomes the efficient cause more particularly when aggravated by losses of albumen, as in amyloid disease of the kidneys. Œdema about the ankles is, therefore, one of the first signs of the renal affection. When both of these causes co-operate, a very considerable amount of œdema may be developed. In the majority of cases, however, this symptom is absent throughout the disease.

Another cutaneous symptom which deserves mention is the alteration in the *soft parts* of the terminal phalanges and in the form of the *nails* of the fingers and toes.

This alteration consists of a bulbous enlargement of the third phalanx, and a curving of the nails, especially in their long diameter, giving them the appearance of claws. The most strik-

ing development of this drum-stick shape, as it is commonly called, is not seen in phthisis, but in those inherited cardiac valvular lesions which are accompanied by marked cyanosis. It also occurs in other chronic diseases of the respiratory organs in a more typical form than in phthisis. The most exquisite examples which I have seen have been in long-continued emphysema with thoracic fistulæ. In a girl, eighteen years of age, who for five years had had a thoracic fistula in the fourth intercostal space on the left side, discharging large quantities of pus daily, these deformities on the fingers and toes had attained extraordinary dimensions. The upper half of the chest was completely collapsed, while the lower part was dilated by the effusion.

In consumption this sign is most fully developed in the very chronic cases, where a considerable portion of the lungs has become impermeable, and it occurs also more frequently in patients who enjoy a fair degree of health and are able to work, than in those who early become bedridden from more active disease.

The only cause to which I can ascribe this symptom is the retardation in the return current of the venous blood, though this is rather a forced explanation.

Anomalies occur also in the growth of the *hair*. In most cases it atrophies, falls out, becomes dry, or turns gray. The *red line on the gums*, which was formerly regarded as a symptom of the disease, certainly does occur in many patients, and at a very early period, but it is also found in other persons. At the same time the enamel of the teeth becomes dissolved, perhaps from the acidity of the buccal fluids, or from some other analogous cause, and decay of the crown is produced.

The *emaciation* and the *fever* are symptoms of such importance that I have intentionally reserved them for final consideration.

The emaciation is the most striking, and the fever the most important symptom of phthisis; from the former the disease has from of old taken its name, while the recent investigations into the nature of the latter have led to discoveries which have materially increased its importance. The emaciation depends only in

part upon the fever, and for this reason the two symptoms must be considered separately.

Emaciation.

In consumption loss of weight is noticed from the very beginning of the disease, and in many cases long before any pulmonary symptoms occur.

The more obscure the origin of this symptom, the more significant is it of the approach of phthisis. It advances *pari passu* with the progress of the disease, and finally reaches as high a degree of development as is ever attained in any affection. The wasting of the body is the most constant indication of the phthisical character of a pulmonary disease. During the periods of remission the patient gains in weight, and may even become heavier than before his illness; but if he begin to lose weight again without assignable cause, a renewed attack may be expected. The number of pounds which may be lost in a given time varies according to the weight of the patient and the relative proportion of fat. The stout woman, previously referred to, had lost forty of her two hundred and forty pounds within four weeks—the sixth part of the weight of her body—and yet exhibited no evidence of pulmonary lesion except the occurrence of hæmoptysis. The total loss produced by the disease is generally from a fourth to a third part of the initial weight. This is the same proportion noticed by Chossat,[1] in the case of dogs who were starved to death. Still, the very rapidity of the loss of weight which is produced within a few days in dogs entirely deprived of nourishment, must necessarily produce many differences between this form of emaciation and that which takes place gradually during the course of phthisis. Trustworthy records of the comparative weights in the same individual, previous to the disease, and during the last days of life or at the autopsy, are very rarely to be obtained. Except in the case of hospital patients it is difficult to observe the accuracy which is desirable, and to avoid the mistakes arising from not making allowances for the clothing, the meals, and the evacuations from

[1] Recherches sur l'inanition. Paris, 1843.

the bowels; and this is the reason why more exact investigations have not been made in regard to the influence which the degree of fever and the amount of nourishment taken have upon the weight of the body in the different varieties of the disease. At all events, the loss of weight must depend upon a diminished ingestion, or upon increased excretion, or upon both together.

Many consumptives lose flesh because they take too little food. In other cases, notwithstanding the ingestion of a sufficient quantity, the food is imperfectly assimilated, and most of it passes away in the excretions from the bowels. This malassimilation may depend upon fatty disease of the liver, disease of the mesenteric glands, or some other lesion of like character.

When diarrhœa occurs, the emaciation proceeds rapidly, even though the appetite be good. The effect of expectoration, hemorrhages, and perspirations, in producing loss of weight, is obvious.

But very often it happens that there is a striking loss of weight, although the patient is unaware of any change in the quantity or quality of his food, or of anything abnormal about his evacuations. In such cases the source of the loss sometimes escapes us, because we have no means of ascertaining what the previous condition of the patient has been in regard to fever. It is possible, therefore, that the remarkable disappearance of fat, which is often noticed even in the early stages of phthisis, may be due to a destructive metamorphosis and excretion of this substance through the lungs, skin, or kidneys, in a way which we have as yet no means of detecting.

The wasting, or loss of weight, involves, however, not merely the fat, but also all the tissues and the blood; but in what different proportions these are affected in consumption cannot be determined in the absence of special reports on this point. Although there are many other factors which serve to explain the exhaustion of the vital forces, yet the loss of weight in all the organs, in addition to the special pathologico-anatomical changes in the lungs, intestines, etc., must be regarded as one of the chief causes of the fatal result. Hence the name consumption is appropriate.

In addition to the above-mentioned causes of the loss of

weight there is still another, and that the most important one, viz., the *fever*. This is not the place to consider the various explanations and experiments which go to show that the fever is due sometimes to an increased production of heat, and at other times to its accumulation in consequence of lessened discharge, or to both causes combined. For the present, however, our only available test for the fever is the elevation of the temperature; and the general proposition holds good that in febrile patients the loss of weight corresponds to the height and duration of this elevation. Experience teaches, also, that the most marked increase of weight occurs during those intervals in the disease when only normal temperatures are found, although in some cases the same result may be produced by special modes of treatment, notwithstanding the existence of slight elevations of temperature.

Although the various forms of phthisis present great differences in the amount of this elevation, and in regard to the relative duration of the pyretic and apyretic periods, the general fact still remains that there is no consumption without fever.

The intensity of the fever, as a rule, increases in proportion to the rapidity with which the local process advances, and the highest temperatures at the beginning of the disease are less than those found towards its termination.

Moreover, there is the greatest diversity in the fever curves, and no one of them can be said to be characteristic of phthisis; nor are the different forms of the disease to be distinguished by the existence of uniform types peculiar to each. The form of the fever does not enable us to decide upon the nature of the morbid process, whether it be pneumonic, bronchitic, or peribronchitic in character, or whether it be a miliary tuberculosis.

During the course of phthisis all the chief types of fever are to be found, and sometimes in the same individual.

There are cases of florid phthisis running their course within from six to eight weeks, in which the *continuous type* is observed. The morning temperature falls but little below 102°; the evening temperature often rises above 104°. The entire fever curve presents a very uniform appearance. Notwithstanding

this continuous high temperature there are usually no cerebral symptoms. If profuse perspirations occur, the minimum temperature may fall to about 100.4°, and the teeth of the curve become steeper, forming the *remitting type*. When the disease runs a chronic course, the physical signs for weeks and months together showing scarcely any extension of the process, the minima are generally normal, or a little below the normal, while the maxima average from 101.3° to 102.2°; *intermitting type, hectic fever*. Longer intervals may also occur, in which the maximum does not rise above 99.5°, showing the absence of fever. During such a period the weight of the body may, under favorable circumstances, be considerably increased.

Very few cases of phthisis run their course with a perfectly uniform movement. Remissions, exacerbations, and intercurrent complications almost always occur, whatever the mode of progress of the disease, whether it be acute from the outset to its fatal termination, or begin in a latent form and develop gradually; hence the impossibility of abstracting from the very numerous curves, which have been taken from phthisical patients indiscriminately, any definite rules for the course of the temperature in this disease.

In these curves every possible variation occurs. Nor does the numerical method of French writers make the subject any clearer; *non numerandæ, sed perpendendæ observationes*.

Thus there may be high and low maxima, high and low minima, subnormal temperatures, and all these for a few days only, or for several weeks together. The maxima rarely occur in the morning or at mid-day, and the falls to the daily minimum take place with very varying degrees of rapidity. The mean temperatures rise or fall gradually; sometimes they increase towards the end of life, but very often they decrease at this time. Death does not occur when the range of the fever is highest, but is generally preceded for a longer or shorter time by the symptoms of collapse.

In the present state of our knowledge of fever in those diseases, which depend upon local changes in individual organs, it may be asserted that, as a rule, the temperature becomes higher, and its average height greater the more rapidly the local

process extends and undergoes an unfavorable conversion into suppuration, caseation, softening, etc.

The most constant and highest elevations of temperature are found in the variety of the disease known as phthisis florida, in which the autopsy reveals extensive infiltration, with caseous masses, numerous broncho-pneumonic processes, and bronchiectases filled with caseous matter. On the other hand, when the pulmonary changes arise insidiously, and remain but slightly developed, or when even extensive disease exists for a long time without making any progress, the temperature is either normal or only slightly elevated, and then generally in the evening.

In the latter case, however, the local changes are probably principally of a cicatricial or cirrhotic character, with little or no broken-down material to be absorbed. Hence, consumption must be regarded as one of the diseases in which the fever is produced by the absorption of substances derived from the local morbid process. The substances which are most febrific are formed by the necrobiosis of inflammatory products and tissues. Aside from complications the height of the temperature must correspond to the amount of these necrobiotic products, and as this varies in different cases of phthisis, so the fever will at times be higher or lower, or entirely absent.

We know, moreover, that the greater the general waste of the substance of the body, the more profound is the exhaustion, and that in febrile diseases, under these circumstances, the temperature maxima and minima draw apart from each other (auseinanderrücken) and the teeth of the curves become more elevated. Hence, in the later periods of both the acute and chronic varieties of consumption, the fever almost always assumes the hectic type, in which the minima show the collapse and the maxima the constantly recurring absorption of pyrogenic matter.

This form of fever is very apt to be accompanied by profuse *perspirations*, which are a serious annoyance to the patient. But, as has already been mentioned, the sweating is not due solely to the increase of temperature, nor does its amount correspond to the height of this elevation. The fever of phthisis is, however, characterized by the unusual abundance of the perspi-

ration, and this is probably an additional reason for the specially deleterious influence of the fever. Profuse sweating rarely occurs without fever, although an elevated temperature is not essential in every case. These exceptions are probably due to individual peculiarities, for in the human body no secretion is subject to so many variations as the perspiration. Some persons have a dry skin under all circumstances, while others become bathed in perspiration upon the slightest cause. These cases are so familiar to every one that it is unnecessary to dwell upon them. The most profuse perspirations, as is well known, occur without fever and without any other indications of disease, apparently as an independent affection of the skin; and it may be that the excessive sweating of phthisis is sometimes to be explained in this way.

Although the perspirations are not exclusively symptomatic of fever, yet in most cases of consumption the fever does induce perspiration, and this may be considerable, even when there is but a slight elevation of temperature. When the cutaneous secretion is abundant under these circumstances it must be regarded as a peculiarity of the fever.

Sometimes the fever begins with a chill like that of intermittent fever, and then, as is well known, often terminates in a very copious diaphoresis. This early subjective symptom of fever so frequently occurs at a particular hour, that the patient and physician suspect malarial fever. Such a complication may exist in some cases, but one must be careful not to be persistent in such an opinion, since in the majority of instances these intermittent forms of fever depend upon the fundamental disease.

Generally there is only a chilliness, a sensitiveness to the slightest exposure to cold, obliging the patient to draw his clothes about him, or to cover himself with a blanket. The hands and feet become slightly cold. Very soon a flush, which is generally very circumscribed, appears upon the cheeks; the face becomes hot, and the lips dry; thirst occurs, the cough grows more frequent, the breathing is accelerated, the languor increases, and the patient lies down, goes to sleep, and perspires.

The circumscribed "hectic" flush of the cheeks, and the glistening appearance of the eyes (dilated pupils) are popularly

regarded as indicative of phthisis, and undeniably so. How it is that the pulmonary disease produces the central flush of the cheeks is uncertain, but that this symptom is directly connected with the affection of the lungs is shown by the fact that it occurs not only in chronic, but also in acute pneumonia, and is generally most marked on the side corresponding to the lung, which is chiefly affected.

Since the fever is often present during only a part of the day, and the physician's visits are generally made in the forenoon, especially in chronic cases, the fever in phthisis may be overlooked, notwithstanding the use of the thermometer. If there be no morning fever, the existence of coldness of the hands, flushed cheeks, thirst, irritable cough, and night-sweats make an evening visit necessary. At the present day, moreover, we can request all our private families to keep a thermometer, and a little instruction will enable the patient or his attendants to supply us with registrations of temperature, which will enable us to form an opinion of the fever, and of the progress it is making.

The statement previously made, that the fever of phthisis presents all possible variations not only in a series of cases, but even in the same individual, is amply verified by the investigations of Lebert,[1] and by the fifty-seven conclusions drawn by him.

The course of the fever in phthisis will naturally be considerably modified by the occurrence of complications. The chief differences which are thus produced will be referred to again under the head of the diagnosis and prognosis.

The fever is increased especially by inflammatory processes, such as pleuritis, pneumonia, and peritonitis, while the extension of the disease to the larynx and intestine has but little effect. It is sometimes diminished by hemorrhages, and this result is, of course, to be expected when collapse is induced by the supervention of some more formidable process, such as pneumothorax or peritonitis from perforation.

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[1] Veränderungen der Körperwärme im Laufe der Tuberculose. Deutsches Arch., XI., 43.

It is to be observed, moreover, that, as in acute diseases generally, so in phthisis, the course of the temperature curve is the true test of the intensity of the disease. The differences in the forms of phthisis and their danger depend not upon the amount of the local lesions, but only upon their nature, and the rapidity of their progress, and when proper allowances are made for incidental circumstances we have no means for deciding upon these points so certain as the frequent measurement of the temperature.

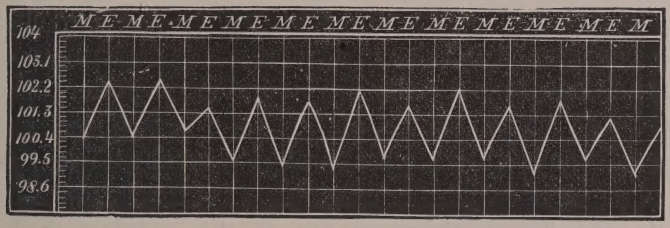
For the purpose of illustrating the principal forms of fever in the different stages and modes of progress of the broncho-pneumonic disease, I give below (page 573) a few specimen curves. The first shows the usual form of fever, the second the hectic form with terminal collapse, the third the beginning of a phthisis florida, and the fourth the end of the same.

Complications.

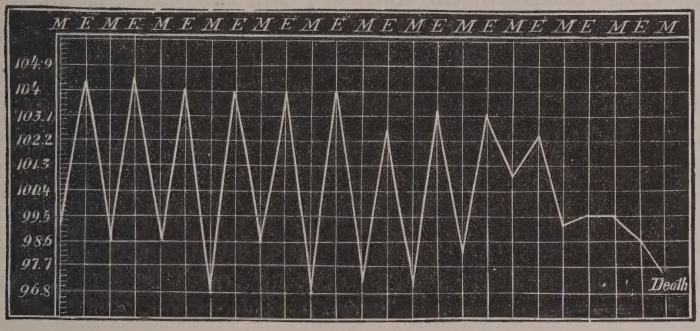
It would be impracticable to consider here all the intercurrent affections which so frequently attack patients during the protracted course of phthisis, or the various illnesses out of which this disease is very often seen to develop; the latter belong rather to the etiology. I shall confine my remarks, therefore, to two groups of affections, one of which is directly connected with the general phthisical process, while the other has hitherto been observed only as frequently coincident with it.

To the first group belongs the formation of secondary miliary tubercules. The acute general miliary tuberculosis will be considered later, and also the allied form of acute tuberculosis, which occurs in the lungs and perhaps other organs during the later stages of phthisis, and is merely one of the modes in which the disease terminates. That this acute tuberculosis is of the nature of a complication is evident from the results of inoculation; but even before these experiments the infectious nature of the affection had been surmised (Buhl). Miliary tuberculosis may, however, occur also in a circumscribed local form; it may spread locally, and give rise to secondary inflammations, which are generally of a chronic character. This view is a very old

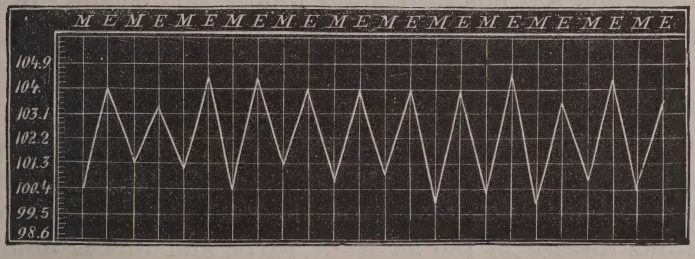
Repeated Attacks of Phthisis during a period of Seven Years, beginning each time with Hæmoptysis. Curve of Chronic Bronchopneumonia.



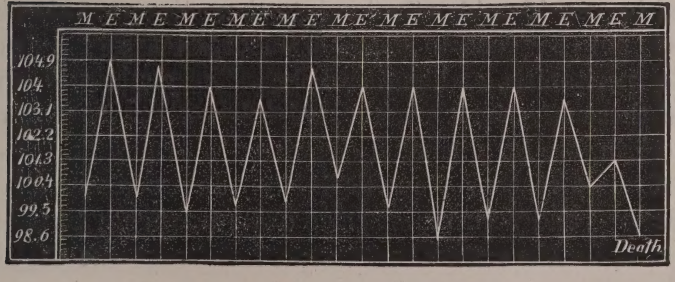
Final Curve of Hectic Fever in a Consumption of Five Months' Duration, which began after Childbirth. Terminal Collapse.



Portion of a Curve in Phthisis florida fatal in Forty Days. First Part.



Curve of Phthisis florida. Second Part.



one (Andral), but its correctness has been demonstrated recently by a series of histological investigations (Rindfleisch, Schüppel, Köster, Friedländer). Whether the frequent pneumonias which occur in consumptives arise in this way it is difficult to decide, because their course is usually as favorable as under other circumstances, as Andral, Louis, and Grisolle have already stated. Indeed some consumptives pass through several attacks; Andral speaks of cases in which the pneumonia recurred from twelve to fifteen times. These observers are of the opinion that pneumonia occurring at the outset of phthisis has no marked influence upon the course of the disease; but the exceptions to this rule are numerous, and are seen especially in those cases in which the pneumonic infiltration occurs in the upper lobes near the phthisical disease. In the later stages of consumption pneumonia not infrequently proves fatal by completing the already existing exhaustion.

In this way, moreover, the tuberculous process extends from the lungs or intestines to the serous membranes, the pleura, peritoneum, and pericardium, producing more or less extensive inflammation of these parts.

In some cases these inflammations result in considerable fluid exudation, and are not recognized unless this occurs; in other instances they remain very circumscribed, their products are dry, and the only indications of their occurrence are the local pain and friction murmurs.

It must not be supposed, however, that these inflammations are exclusively due to a preceding eruption of tubercles; an inflammatory irritation may emanate from any kind of parenchymatous disease in the adjacent parts, and this is doubtless the explanation of the more acute forms of inflammation attended by considerable effusion into these three serous sacs.

This is not the place to consider in detail the symptoms of pleuritis, peritonitis, and pericarditis. The acute and chronic forms of these affections, whether of tuberculous or non-tuberculous origin, are fully treated elsewhere in this work.

For obvious reasons pleuritis is the most common of these complications, and peritonitis is also quite frequent; but pericarditis is rare, for the reason that in this membrane the conditions

are very unfavorable for the occurrence of an eruption of tubercles, or for the action of an inflammatory irritation.

That *meningitis* may also occur in the same way is unquestionable. Here, however, there is occasionally an intermediate factor in the form of antecedent tuberculosis of the brain substance, or of the cranial bones; and then the inflammation may be either diffuse, with copious effusion, or circumscribed, with local formation of miliary tubercles. In these cases the meningitis becomes the most important part of the complication, as is shown by the course of the cerebral affection in children in whom these two causes—tubercle of the brain or cranial bones—are much more frequent, and in fact not very uncommon. Probably every physician who has been in practice a few years has seen such cases in children terminate with cerebral symptoms of a meningitic character.

Each of these four inflammatory complications may develop either with definitely marked symptoms or very insidiously. If there are no constant local pains, aggravated by pressure, to guide us, the existence of the complication will be discovered only by the increasing effusion. When the morbid changes are at all severe, the fever, which has hitherto been remittent or intermittent in character, will always be found to have assumed a continuous type, although perhaps only for a short time. The nearest approach to a cure is seen in the case of the pleuritis. This is the complication also which most frequently runs an entirely latent course. In its more severe forms, however, it produces, as the other complications always do, a rapid aggravation of the disease.

An important characteristic of the pleuritis of consumption is its frequent tendency to attack the two sides of the chest alternately. This is to be accounted for by the fact that, as the phthisical process is rarely confined to one lung, the extension of the irritation to the pleura, and the pleuritis excited thereby, will occur now on one side and now on the other. This mode of occurrence is significant. Should the pleuritis take place before the pulmonary affection can be detected, one may be doubtful as to the etiology; but if the complication recur frequently and alternately on the two sides, the existence of phthisical disease

in the lungs is in a high degree probable, a proposition already laid down by Louis.

Sometimes, though more rarely, the *peritonitis* of phthisis may also abate, and at the autopsy the process may be found to have been completely arrested. In the diffuse forms of inflammation the effusion is usually very abundant, and this, together with the coexisting meteorism, produces a considerable tumefaction of the abdomen, so that the abdominal symptoms are the chief source of complaint. Still, with the exception of tenderness on pressure in various places there is generally little or no pain. In some instances the effusion continues to accumulate; in others the peritonitis is circumscribed, and perhaps limited to the situation of the intestinal ulcers; under these circumstances certain parts are resistent, and frequently present the shape of the intestinal convolutions both on palpation and inspection. Whether general or partial, this tuberculous peritonitis is always chronic; and although chronic peritonitis sometimes occurs without tuberculosis, yet the tuberculous form is by far the most frequent.

Pleuritis and peritonitis, and in very rare instances pericarditis also, may occur, however, in consumption in an entirely different way. They may arise as acute affections produced by perforation of the lungs and intestine in consequence of tuberculous softening of the serous membranes.

Perforation of the pulmonary pleura results in *pneumothorax*, which in its turn excites an acute diffuse inflammation of both pleural surfaces. Aside from the cases in which pneumothorax is produced by wounds of the chest, pulmonary consumption is by far the most frequent cause of this affection.

The perforation, as a rule, occurs in connection with the more advanced forms of the pulmonary affection, and with those stages in which the destructive process is developed more rapidly. In the more gradual forms this accident is usually prevented by adhesion of the pleural surfaces, and hence it rarely occurs in the case of old cavities. Sometimes, however, pneumothorax takes place very early in the disease from the breaking down of deposits, which may be minute in size, situated immediately underneath the pleura. I have seen cases in

which pneumothorax seemed to be the first symptom of the disease; in one of them the complication disappeared without the production of much effusion, and unmistakable pulmonary phthisis developed subsequently. In most cases the pneumothorax continues until death, and generally materially hastens this event, which is not long postponed; still I have met with two patients, who lived one and a half and two years respectively, the affection being finally converted almost entirely into empyema.

As the rupture of the pleura, and the escape of air and more or less irritating substances into the pleuritic cavity take place in a moment, the symptoms are always very acute. The patient generally feels a severe stabbing pain in the affected side, which seriously interferes with the expansion of the chest. Still more marked a symptom is the dyspnœa, which results from the sudden diminution of the respiratory surface. As the affected lung is still capable of contraction, every expiration, as well as inspiration, drives air through the opening into the pleural cavity, which thus very soon becomes filled with air in a state of tension, and thus considerable displacement of adjacent organs is produced. The mediastinum, together with the heart, is driven far over towards the healthy side, the intercostal spaces are broadened or prominent, the diaphragm is pushed convexly downwards into the abdomen, and can be felt below the ribs as an elastic crescentic swelling, while the ribs retain their inspiratory expansion, because they are no longer drawn downwards by the tractile elasticity of the pulmonary parenchyma.

In consequence of these rapidly developed and important changes, accompanied as they must necessarily be by a serious interference with the pulmonary circulation, the heart's action becomes very irritable, the pulse extremely rapid, and the pale face and nails very often cyanotic, from obstruction to the return flow of venous blood into the right heart. The extremities become cold, the intense dyspnoic muscular exertion produces a profuse perspiration, and in many cases the general collapse is the forerunner of speedy death. At other times the patient recovers from the shock, the breathing becomes more quiet, the pulse regains its force, and loses its frequency. Pleuritic effu-

sion now develops, always suppurative in character; it usually increases, and finally, after displacing the air it almost completely fills the pleuritic cavity. Sometimes the patient so far regains his strength as to go about, and if the case be now seen for the first time, the original occurrence of perforation and the phthisical nature of the disease may still be ascertained by careful inquiry into the history of the case.

An effusion of this kind, whether preceded by perforation or not, compresses the lung completely, drives the blood from its tissues, and in this way arrests the further progress of the phthisical changes. It is therefore either the pleuritis itself, or the advancing disease in the other lung combined with it, which consumes the patient's strength.

Acute peritonitis may arise in a similar way by perforation of the peritoneal coat of the intestine. Here also the escape of gas and other contents of the bowel excites a violent inflammation, which is only in rare instances prevented from spreading by the previous agglutination of the abdominal viscera. The intense pain, which rapidly ensues, is accompanied by nausea or vomiting, and is followed by speedy collapse, so that the signs of effusion are usually not well developed. The abdominal wall is more tense than swollen, and is everywhere very sensitive to pressure, while the urine is partly suppressed, and is discharged in small quantities with painful urgency. In the recumbent position of the patient the percussion note over the region of the liver is tympanitic, in consequence of the organ's being pushed backward by the gas, which accumulates in the peritoneal cavity, and naturally seeks the uppermost position.

The rapidity with which the fatal collapse is developed depends upon the degree of enfeeblement already existing. The asphyxia, which ultimately becomes complete, manifests itself by the same group of symptoms which uniformly characterize all severe irritations of the peritoneum, and announce the certain and impending end, viz., the Hippocratic countenance, the whispering voice, the rapid thoracic respiration, the cold cyanotic extremities, and sometimes the occurrence of muscular spasms.

Peritonitis from perforation occurs, however, in phthisis

much more rarely than pleuritis from the same cause, and in the majority, though not a very large majority, of cases this form of peritonitis proceeds from other causes than ulceration of the intestine. But here, as in other cases, it sometimes happens that the occurrence of perforation is sometimes the first indication of the existence of the ulcerative process.

In very rare instances acute *pericarditis* has also been observed in connection with the rupture of a cavity or a softened tuberculous mass into the pericardium.

Among the complications which are not produced directly by the phthisical process itself, but which give to the disease a special character, there still remain to be considered certain affections of the *digestive organs* and the *amyloid degeneration* of the *liver*, *spleen*, and particularly the *kidneys*.

It has already been mentioned in the symptomatology that in a certain number of patients symptoms of gastric derangement manifest themselves so early, and assume such a preponderance, that they seem to constitute the chief part of the disease, the pulmonary affection being discovered only by direct examination.

Loss of appetite, nausea, occasional vomiting, thirst, and tenderness in the epigastrium, with or without furring of the tongue, sometimes occur so early that it is supposed that we ought to regard them as causes of the pulmonary disease. At all events, in whatever form and at whatever period of the disease these symptoms are developed, they are a very unfortunate complication, because they interfere seriously with the treatment.

The only anatomical changes in the mucous membrane of the stomach thus far discovered to account for this complication is the gastric catarrh. This condition, it is true, presents the same symptoms, but the term gastric catarrh is used to cover too wide a field in the pathology of gastric affections. As to the more or less extensive softening of the mucous membrane, which was affirmed by Louis to be the morbid condition found in this complication, it must be dismissed from consideration, because it is no longer regarded as an ante-mortem lesion, and, even if it were, it would not explain the symptoms any better. On the

other hand, in many cases where the complications in question continue to the last, we find the mucous membrane described as presenting the same appearances as in other cases which are unattended by gastric symptoms ; that is, as congested, anæmic, thickened, thinned, covered with mucus, etc., or as entirely free from signs of disease.

But I have already admitted the great difficulty of ascertaining the pathologico-anatomical condition of the gastric mucous membrane, and especially of explaining its symptoms. These, as well as the functional disturbances of the stomach, are undoubtedly dependent upon many other things besides the permanent anatomical lesions, as daily experience shows; in fact, the prospect of a satisfactory explanation of the causes which underlie the gastric complication in individual cases is for obvious reasons anything but flattering.

The symptoms themselves, as they occur in phthisis, do not present any special character.

The most frequent symptom is a lack of appetite, a distaste for most articles of food, especially those which are nutritious, sometimes a loathing at the very idea of food, and finally vomiting, when nourishment is forced upon the patient. The other symptoms of catarrh may be entirely absent, nor are there necessarily any acid eructations or cardialgia, such as one delights to hear complained of in order to be able to use the bicarbonate of soda. On the contrary, there is often so much anæmia, and so many indications of disturbed innervation in other parts of the nervous system, that one is inclined to regard this dyspepsia also as essentially nervous in character. To what extent these symptoms are influenced by coexisting affections of the liver (fatty or amyloid degeneration) has never been investigated, and must be reserved for future inquiry.

Amyloid degeneration of the abdominal organs occurs as a complication in consumption, just as it does in various other profound constitutional diseases, and for this reason chronic phthisis is generally supposed to be one of its causes, although the connection between them is not very clear. That there is some connection, however, is well established by clinical experience ; also that the complication does not occur until after the

pulmonary disease has lasted for some time, and belongs to those forms of phthisis which run a chronic course. Its appearance is generally preceded by the existence of cavities of various sizes, and more or less copious suppuration. Meckel, in his remarkable work on this subject, says that "the tuberculosis changes suddenly into the amyloid disease," and he gives cases in which a recently "healed tuberculosis" is noted along with the amyloid disease of the organs. Such a connection between the two affections has, however, never yet been proved to exist. The symptoms of the pulmonary disease, it is true, often abate, but the intestinal ulcerations still continue, and the cases are not uncommon in which the autopsy shows that even the tuberculous processes also exhibit indications of recent extension and change.

However this may be, the amyloid disease exerts a detrimental rather than a favorable influence upon the course of the disease, and seems rather to shorten life.

The amyloid disease in pulmonary consumption attacks the spleen, liver, kidneys, and intestinal mucous membrane; the affection making its appearance first in the spleen and liver, as it does under other circumstances. These two organs are undoubtedly connected with the formation of blood-cells, and this function must, therefore, from the start be impaired by a disease which usually invades their whole structure; hence, unless the spleen and liver have been already found by physical examination to be increased in size, the first symptom noticed will be a very evident diminution in the red blood-corpuscles, and a consequent pallor of the skin and mucous membranes. As the amyloid enlargement of these organs seldom occasions any local pains, the pallor and the physical signs constitute the only manifestations of the disease. But when the *kidneys* also become involved, albuminuria occurs, and, before long, dropsy; and as all these changes are very often present in consumptives, who are still able to go about and perhaps to attend to their business, not infrequently the first sign of the complication which attracts attention is the occurrence of œdema about the ankles. The albuminuria is usually so considerable, and the treatment so ineffectual, that the dropsy rarely disappears entirely. The lat-

ter is, however, rarely excessive in amount, because the patient's strength now fails rapidly, he takes to his bed, and generally succumbs in a short time. The frequency with which the intestinal mucous membrane becomes implicated in the amyloid disease cannot be determined ; I know of no statistics on this point. This complication manifests itself during life only by diarrhœa, which, however, is not characteristic ; and as the same symptom may arise in phthisis from other causes, even its obstinacy does not justify us in referring it in a given case to amyloid degeneration of the intestinal mucous membrane.

The diagnosis of the amyloid complication during life rests, therefore, upon the pallor and œdema of the skin, the painless enlargement of the spleen and liver, the presence of albumen in the urine, and the other properties of this secretion which contraindicate an inflammatory nature of the renal affection ; symptoms which are, in fact, a sufficient guide.

The symptoms on the part of the respiratory organs sometimes diminish, but not to a striking degree, and very often, although the cough and expectoration abate, the appetite declines at the same time ; and even the patient himself, with his œdema before his eyes, cannot help seeing at he is failing, not to mention the incessant diarrhœa.

Diagnosis.

The term consumption of the lungs, whether used in its strictly literal signification, or even scientifically, is merely a general expression for the common result of a variety of forms of disease in the respiratory organs. The initial stages of none of these forms necessarily lead to phthisis, but each of them may do so ; in some this result almost always follows, in others only occasionally.

Moreover, these different forms are very rarely met with unmixed ; one form passes into the other ; and although for purposes of study we separate them from each other, yet they are all to be regarded as the same disease, as merely different modifications of the course of the pulmonary phthisis.

When, however, we come to speak of the diagnosis, course,

and prognosis of the disease, this general mode of treatment is unsatisfactory; it is better to separate the morbid process into its chief types, only we should bear in mind that the latter are very often met with in combination or in succession in the same individual.

I have already pointed out the fact that the *acute* miliary tuberculosis is a special affection, which cannot be classed with pulmonary consumption, from which it differs widely not only in the rapidity of its course, but also in its symptoms and in the mode of death. When, however, it occurs as a complication of phthisis, its influence in producing the fatal result will have to be considered; but here, in our description of the individual forms, it may be omitted.

As to a *chronic* miliary tuberculosis, it can hardly be supposed to have an existence, if we have correctly interpreted the origin of the miliary tubercle. Since a large portion of the small, more indurated nodules are dissolved in the broken down inflammatory products, and since investigation has shown that the miliary tubercle undergoes but *one* change, viz., death, the existence of what has been characterized as persistent miliary tubercle must at the present day be denied; consequently it can no longer be maintained that there is a form of pulmonary consumption produced by this chronic miliary tubercle, or that it is possible to diagnosticate such a condition.

There remain to be considered also the various forms of *inflammation*, which differ from each other, partly in their extent, partly in their metamorphoses, and partly in the tissues which they occupy. According as one or the other of these forms predominate, the phthisis will be variously modified, and corresponding types of the disease will be produced which can be distinguished from each other.

The clinical separation between the various forms of pulmonary consumption has as yet been imperfectly effected. We require a closer study of the full history of cases in connection with the autopsy, and the latter should include more than the mere microscopical examination of sections of organs. In this way only shall we be able to regain a position where, with better material, we can carry out the general method which

characterizes the brilliant period of French clinical medicine in this century, and which secures to the works of Laënnec, Louis, Andral, Grisolle, etc., an enduring value. From a sufficient number of such cases, well observed during life and carefully examined after death, microscopically and otherwise, we shall gradually be able to formulate the necessary rules upon which a correct diagnosis can be based.

This being premised, it seems to me that for the present we may regard the different varieties of phthisis as dependent upon three forms of pulmonary lesions and their combinations with each other.

All three of these forms, it is to be understood, begin in the *upper lobes*, or *apices of the lungs*, and extend downwards.

The *first* form is the *simple chronic pneumonia of the apex*, in which the changes consist only of simple chronic indurative inflammation.

The *second* form is represented by the *broncho-pneumonias*, in which the first change is the formation of clusters of nodular foci, which excite inflammation in the surrounding parts, and coalesce with the inflammatory products thus formed. The whole mass then becomes caseous, and breaks down in numerous places, forming ulcerating cavities, and thus the destructive process may steadily advance until it extends beyond the upper lobe, or it may be arrested by the occurrence of simple inflammations resulting in induration.

The *third* form consists of morbid processes, which begin in the *bronchi*, and there produce inflammatory infiltration and swelling of the entire thickness of their walls, with caseation and dilatation. Ulcerative destruction then occurs, pneumonic processes are set up by the extension of the disease into the parenchyma, and the inflammatory products thus formed either become necrosed and break down into caseous matter, or undergo induration resulting in limitation and cure.

The *diagnosis of the first form*, the simple chronic pneumonia of the upper lobe, is based chiefly upon the physical signs.

The most important sign is dulness in the supra- and infra-spinous fossæ, and in the supra- and infra-clavicular regions. The more intense and extensive this dulness, the greater the

probability that this is the form of the disease. At a later period, however, the altered shape and the impeded respiratory movements on the affected side show that shrinking has supervened. The sinking above and below the clavicle, and the flattening of the whole upper region of the chest belong to chronic pneumonia. On auscultation the respiratory murmur is always found to be abnormal; it changes gradually from a prolongation of the respiratory sound to a loud, harsh, bronchial sound heard both on inspiration and expiration. Crackling râles may be heard at times, but they are often entirely absent. When they do occur, they are sibilant in character. If catarrh be also present, the râles are sometimes abundant, but they soon diminish very much in number, or disappear entirely.

These local signs either very slowly extend, or as gradually become confined to a smaller region, where they remain permanently fixed. If bronchiectases are produced by retraction of the connective tissue, signs of cavities of corresponding size will be found.

The cough is usually of moderate severity, or may be absent; the expectoration is scanty, but in the case of intercurrent bronchial catarrh it is more abundant, always muco-purulent, and merely catarrhal in character.

The sputa contain no blood, and hæmoptysis does not belong to this form. Pains, which are supposed to be rheumatic, not infrequently occur on the affected side.

The functions of other organs are normal; there is either no evening fever, or only an inconsiderable amount, and the emaciation, which is at first slight, increases slowly, or may almost entirely disappear.

In many instances there is no evidence of heredity, but the history of the case shows the existence of a local cause, such as a pneumonia, from which the patient has recovered, or a wound, or the effect of particles of irritating dusts, as in the chronic apex pneumonias seen in operatives.

The *lobular disease characterized by foci*, the broncho-pneumonia, differs very materially from this description.

In this form symptoms are noticed before any physical examination is made.

The patient at the outset has a dry cough, or a hemorrhage occurs, which is followed by little or no expectoration. Sometimes, however, these symptoms are preceded by emaciation, paleness, derangements of the appetite, and fever, which may be accompanied by night-sweats. The majority of such patients are young persons of phthisical parentage, who have grown rapidly, and have stunted chests, or who have recently passed through measles, whooping-cough, typhus, or childbirth. In childhood they have suffered from scrofula, and later in life have had several attacks of pneumonia or pleurisy.

When expectoration occurs, the sputa frequently contain gelatinous admixtures of a slightly red color, and quite early in the disease elastic fibres. At a later period they become globular, solid, and partly purulent, with more or less blood at different times.

Fever in various degrees is always present, accompanied by perspiration during sleep. In most cases the specific lesions of the larynx are also superadded, and the intestinal functions are rarely undisturbed. The emaciation advances *pari passu* with the fever and the complications, and as the patient is weighed from week to week the disease is found to be steadily progressing.

The physical examination shows that the upper parts of the chest are narrowed, and expand imperfectly, often unequally on the two sides. The supra-clavicular fossa is sunken, there is slight dulness, the apices of the lungs are lowered in position, and when the destructive processes are further advanced there will naturally be found tympanitic resonance, or the bruit de pot fêlé.

On auscultation the signs of infiltration predominate over those of bronchitis. The crackling râles are scanty at the outset, but later, when softening occurs, they become more abundant, and mixed with sibilant bubbling. The expiratory sound is usually bronchial, and in many cases the inspiratory sound has an interrupted vesicular or uncertain character for a long time before bronchial inspiration is heard. As the disease progresses auscultatory signs of cavities make their appearance.

The *third form* appears at first as a *catarrh of the apex*, but,

as in the preceding form, there are usually early constitutional symptoms, such as slight fever, loss of strength, pallor, and emaciation. The first symptom is cough, with expectoration, which frequently contains streaks of blood. Non-sibilant crackling râles are to be heard from the beginning of the disease, at least on deep inspiration or coughing. The position of the apices above the clavicle remains about normal, and dulness cannot be detected. The sputa soon contain yellowish-white, non-aerated streaks, and much amorphous matter, together with small white detached particles, which sink in water to the bottom.

This form, like the preceding one, affects scrofulous persons, who inherit a predisposition to phthisis, and have frequent attacks of catarrh; in fact, they assert that they are constantly catching cold. The crackling râles extend over a larger surface, and the first shades of dulness develop very gradually in the apices, but sometimes also a little farther down, and some of the râles become sibilant.

The laryngeal and intestinal symptoms make their appearance later than in the previous form.

It is evident from the description of the second and third forms that there is no sharply defined distinction between them, and that for this reason the differential diagnosis is by no means easy. The difficulty is still further increased by the fact that they often occur in connection with each other at an early period of the disease, and sometimes from the very start.

A similar difficulty presents itself in regard to the distinction between the *simple* chronic inflammatory changes, and the *specific* broncho-pneumonic, or bronchitic processes, which produce caseous material, and which may be briefly described as scrofulous or tuberculous.

These forms, as has been repeatedly mentioned, very often occur in combination, and hence in the diagnosis of phthisis it is not always possible in a given case to refer the disease to the special variety to which it belongs. In such cases we must rely upon the general fact common to all of these varieties, that the phthisical nature of the morbid process is to be inferred from its localization in the upper lobes.

The physical signs by which these lesions can be detected have already been described in the symptomatology, but it should be again insisted upon that the existence of crackling râles, or the slightest abnormity in the percussion note, or respiratory murmur in the apex, provided they are heard constantly or frequently, are significant, especially if they become clearer, or more wide-spread after repeated examinations.

Reference has always been made to the fact that a prolongation of the expiratory sound, and an increase of the vocal resonance above and below the *right clavicle*, and in the *right* supraspinous fossa are met with even in healthy lungs, and that these signs are insufficient by themselves to indicate disease. This point has already been discussed in detail by Louis.[1]

But even when no physical changes can be discovered, the altered shape of the upper parts of the chest, their defective mobility, and the failure of one side to expand equally with the other on inspiration excite serious suspicion. This is especially the case with persons who exhibit an hereditary tendency to the disease, and who have had hæmoptysis, which cannot be otherwise satisfactorily explained. Phthisis may, moreover, be strongly suspected when a patient with cough has had, or still has, a *protracted* and *uncontrollable diarrhœa*, or when a long-continued aphonia is found to depend upon ulcerative, *non-syphilitic*, laryngeal disease, especially when the latter is unilateral, or when *fever* is present without any other obvious cause.

Although the diagnosis of consumption becomes certain only when apex disease is found to coexist with progressive emaciation, yet even when no signs of wasting can be detected, every affection located in that situation makes it probable, or at least awakens our apprehension, that the case will ultimately terminate in genuine phthisis.

So also *emaciation* or loss of weight is very significant when it occurs without any diminution in the amount of food taken, and without recognizable increase in the discharges, particularly without diarrhœa. Whether such loss of weight is always

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[1] L. c., p. 531.

accompanied by an appreciable degree of fever has never been determined, but this is most probably the case. Thermometry is a science of too recent date to have settled this point. A fever, which arises without any discoverable local affection, or other obvious cause, which conforms to no definite type, and lasts longer than any known form of fever, points with great probability to phthisis. On the other hand, if there be an entire absence of fever, and both apices be found to be normal, the diagnosis of phthisis may be dismissed, however suspicious the constitutional symptoms. These general rules are of great service in the *differential diagnosis* between incipient phthisis and anæmia, combined with dyspepsia and hysteria. In the latter affection the pallor of the integuments may be accompanied by persistent cough and emaciation, but the thermometer decides the question. Absence of fever contraindicates phthisis. The same guide serves us in those cases of obstinate tracheal catarrh, or, more correctly speaking, hyperæmia without secretion, which occur especially in women, and excite apprehension of commencing consumption; also in the various forms of nervous cough to which women are particularly subject. As regards *chronic bronchial catarrh*, difficulty can arise only when its signs are present at the same time in the apices. In these cases the diagnosis turns upon the fact, that in catarrh the signs are most marked in the lower portions of the lungs, and that as improvement occurs the signs disappear first in the upper lobes. Unless acute catarrh, with fever, arise as a complication, and then the course of the affection will immediately apprise us of its occurrence, the absence of fever is in favor of catarrh.

In the *later stages* of the disease, when the local affection is well developed, the physical signs and the history of the case will be sufficient to enable us to discover whether the predominant symptoms indicate a simple chronic or a specific inflammation, and here also the presence of fever is an important guide. If there be unmistakable apex disease, with entire absence of fever, the case is either a very mild form of simple chronic pneumonia, or one in which the entire affection is for the time being arrested. The same inference may be drawn if there be a steady gain in weight, because such a gain is hardly consistent with a

continuance of the fever. Sometimes we may be assisted also in the diagnosis by finding that the spirometrical, and perhaps the pneumatometrical, results show an increased activity of the respiratory function.

Although persons with an hereditary, or acquired predisposition to phthisis not infrequently exhibit the phthisical habit of body which may then be regarded as a premonitory indication, or even as a first symptom of the disease, yet this is by no means the rule; cases constantly occur in which, at the outset, nothing abnormal is noticed in the shape of the chest, or in the respiratory muscles, nor are there any indications of that general alteration in the aspect of the patient, which it is almost impossible to describe. But whatever the form of the disease, the farther it advances the more distinctly developed become these general characteristics of the phthisical habit; and although it would be extravagant, and would lead to serious mistakes, to affirm that it is only necessary to have a consumptive patient thrust his hand in at the door in order to recognize his disease from his nails (*ex ungue leonem*), yet as a matter of fact the appearance of a consumptive in the later stages of the affection is so characteristic that with ordinary skill and experience the diagnosis can usually be made at once, without a physical examination. It is unnecessary to dwell upon this point; no description will be of any avail to one who neglects the use of his own senses.

The *sputa* also furnish us with valuable diagnostic indications. At present it would be hazardous to diagnosticate phthisis from the sputa alone, but I am confident that the microchemistry of the expectoration will some day disclose to us substances which are characteristic of the caseous process. The detection of elastic fibres, in connection with some few other data, of course makes it certain that destructive changes are going on in the lungs. This sign belongs, however, especially to the later stages. To the experienced physician the general form of the sputa, which consist of solid, globular, lumpy masses, of constantly increasing size, is, like the expression of the face, almost pathognomonic. There is, to be sure, no positive evidence that these masses when lying in the respiratory organs

had the same shape which they present after expectoration, or that they have come from pathological cavities, but this supposition is rendered highly probable by their non-aerated condition, their similarity in form, and their comparatively solid consistence. Many persons ascribe importance to the uneven surface and corroded appearance of the sputa, but I am very doubtful whether we are authorized in carrying to such an extreme the idea that these sputa are plastic casts of the spaces they have occupied. In all cavities lined with a very vascular connective tissue, which is often covered with pavement epithelium, pus is probably always formed whenever an inflammatory irritation is present; and the amount of pus will correspond to the extent of the neoplastic secreting surface and the intensity of the irritation. Consequently, when the sputa are unmistakably purulent, we may suspect the existence of cavities and may form some idea of their size.

Allusion has already been made in the symptomatology to the diagnostic importance and ominous significance of the slate-gray and reddish purulent sputa.

The tuberculous laryngeal ulcerations which occur late in the disease, like those of the earlier stages, indicate the existence of broncho-pneumonic processes rather than simple pneumonia, and the same may be said of the diarrhœa, if it be dependent upon ulcerations, as shown by its obstinacy and by local pains at various parts of the abdomen. Pneumothorax, with rare exceptions, indicates advanced phthisis. Peritonitis from perforation also belongs to the later stages, and points to the presence of an intestinal ulcer, unless this complication happen to be connected with a round ulcer of the stomach. In this case the situation where the sudden abdominal pain is first felt will generally be a sufficient guide.

Enlargement of the liver and spleen, and albuminuria, associated with the symptoms of amyloid disease of the kidneys, also indicate advanced disease.

Course and Termination.

Our consideration of the different forms of pulmonary con-

sumption has shown, and in fact universal experience testifies, that the course of the disease varies within extraordinarily wide limits. Sometimes, as in the so-called acute cases, which are always of a specific broncho-pneumonic character, the disease proves fatal in a few months; in other cases, the morbid process is arrested, although residua of considerable extent, cavities, and extensive shrinking can still be detected, and the patient bids fair under favorable circumstances to live the usual duration of life. The former variety of the disease is common enough; the latter much more rare. Still, all extensive changes in important organs, even though the lesions may have been anatomically healed by cicatrization, necessarily impair health and shorten life.

It is only in these *acute forms* that the disease runs its course to the end in a *single attack*, without any important remission, and with high fever, early hectic, and rapid emaciation. In the *great majority* of cases of phthisis the symptoms differ at different times, and the course forms a curve with exacerbations and remissions of varying degree.

The *simple chronic pneumonia of the apex* very often begins with symptoms of an ill-defined character, which continue for a time and terminate in a long period of arrest, which frequently lasts for years. Then a second attack occurs, usually on the opposite side, and perhaps the only information that can be obtained in regard to the probable duration of the disease is derived from the condition of the old process. In this way several more attacks may follow each other; the intervals between them generally becoming shorter and shorter, the average health during the intervals more and more impaired, and the emaciation, the slowly advancing local symptoms, the more abundant expectoration, the lowered respiratory energy, and the increased shortness of breath on exertion, showing too plainly that the patient's vital powers are failing. The duration of life in these cases probably depends chiefly upon the strength and vital conditions of the individual, and upon the age at which the pneumonic processes make their first appearance. Life insurance companies reject all applicants who exhibit signs of chronic pneumonia, however slight.

Now and then, however, cases occur in which, after a single attack or only a few repetitions, a permanent cure seems to be effected; the patient regains his flesh, and, notwithstanding the local damage done by the pulmonary disease, the functions of the body are carried on as well as before.

Chronic pneumonia may also be healed permanently, or, as is more commonly the case, after several relapses and gradual advances, the disease terminates in a premature marasmus.

These simple contrasts to the acutely fatal form are, however, exceptional.

Usually the later attacks of the disease no longer present this simple character; they begin with a more intense fever, a more rapid enervation, hemorrhage, and signs of more extensive caseous destruction, and either terminate fatally or leave the patient in a state of great prostration. Should a limitation of such an attack of tuberculous broncho-pneumonia occur, the fever diminishes, or perhaps gradually disappears entirely; the sweating abates, the expectoration becomes more purulent and easier, the physical signs indicate more or less clearly the existence of cavities, the pleuritic pains cease, and the patient gains in strength and weight. Even in these cases also the disease may be arrested by the occurrence of caseous destructive processes, which prevent further infection and tuberculous inflammation of surrounding parts.

A recurrence of such a broncho-pneumonic attack, accompanied as it often is by severe hemorrhages from previously formed cavities, the vessels of whose walls may have shared in the inflammatory irritation, marks another step in the downward progress; and finally, in one of these attacks, the limiting cirrhotic inflammation no longer takes place, and fatal exhaustion ensues. This, which is the most common course of the disease, may also last for years.

When, however, the disease begins not with a chronic pneumonia, but with an inflammation, which is tuberculous from the start, and the very first attack of which is characterized by a more rapid emaciation and more intense fever, the total duration is shorter. These forms are seen in young persons, in whom several causes co-operate, such as heredity, scrofula, acute febrile

diseases, measles, typhus, and the parturient state. In these cases the intermissions are more incomplete, the relapses follow each other more rapidly, and fewer of them are required to produce the fatal exhaustion.

This result is hastened also by the occurrence of *diarrhœa*. As soon as this symptom makes its appearance, especially if it depend upon an actual migration of the tuberculous disease to the intestinal mucous membrane, a more rapid prostration ensues. The loss of weight and marasmus in these cases depend upon the increased intestinal discharges, and are unattended by an elevated temperature; in fact, the temperature may even be subnormal; hence the teeth of the fever curve become very long without the peaks being particularly high.

The same effect is also usually produced when the anæmia is increased by *amyloid disease*, and when œdemas occur in consequence of albuminuria.

In both of these cases, as has already been mentioned, the symptoms of the pulmonary affection ordinarily abate, the cough and expectoration diminish, and in the case of the amyloid degeneration an anatomical arrest, a demonstrable process of cure may take place in the lungs, although the fatal event is thereby but little, if at all, delayed.

Repeated *hemorrhages* act in the same way. Although instances occur now and then in which hæmoptysis recurs frequently in cases where a simple chronic pneumonia has been arrested, or apparently cured, yet, as a rule, these accidents have an unfavorable influence upon the course of the disease. In these less serious forms the hemorrhage undoubtedly always comes from vessels so situated in the walls of cavities that bleeding is liable to occur from time to time. In these cases the loss of blood may be so profuse, or so frequently repeated, that death results directly from this cause, without the intervention of increased destructive changes in the lungs.

This is the usual variety of pulmonary hemorrhage, but in other cases the bleeding may occur as a symptom of a renewed attack of the disease, or it may favor the extension of the same, and for these reasons the occurrence of this symptom is an unfavorable indication.

The detrimental effect of *perforation* of the lung or intestine upon the course of the disease is still more striking. As has already been mentioned, the patient may recover from the former accident, or he may linger for years with an empyema; but these are exceptional cases, and do not invalidate the general rule that pneumothorax is the beginning of the end. This is especially true of peritonitis from perforation, which runs its course very rapidly, in fact within a few hours.

In a certain number of cases the hitherto slow progress of the disease changes quite suddenly to a more rapid one. The fever becomes continuous, the respirations more frequent, symptoms of cyanosis make their appearance, the pulse becomes very rapid and small, the patient usually lies in an apathetic state, and a generally diffused bronchial catarrh can be detected on auscultation. Sometimes the spleen becomes somewhat enlarged, and death results within a few weeks. At the autopsy numerous miliary tubercles of larger or smaller size are found between the lesions of older date, especially in the vascular lower lobes; also in many cases in the spleen, liver, kidneys, and serous membranes. The final stage was an acute miliary tuberculosis.

Finally the course of the disease may be brought to an end still more rapidly by an œdema of the lungs, which may, in its turn, depend upon fatty degeneration of the cardiac muscles. This is especially apt to occur in the consumption of drunkards.

Prognosis.

The description already given of the different modes of progress observed in consumption shows that the prognosis must be indefinite. At the same time it is not necessarily bad, as is evident from the not infrequent cases in which a complete cure is effected; that is, in which cicatrizations, often of considerable size, with or without cavities, and with mortar-like concretions are found after death, or in which quite extensive condensations or cavities are detected during life in one or both lobes, although the patient may be said to enjoy perfect health. But these views are so old as to be quite out of fashion nowadays in this period of restless agitation on the subject of consumption, and

yet it seems to me that the tone which our younger writers assume, as if by their arduous labors they had attained a position which entitles them to ignore the merits of such men as Bayle, Laënnec, and Louis, and to look down compassionately on those of us who take any notice of such antiquated greatness, may be explained by the simple fact that they have never read the works of these authors.

The cure of phthisis is very fully treated by Laënnec.

My previous remarks upon the course of the disease, as modified by its dependence upon a chronic inflammation, or upon a specific broncho-pneumonia, or upon the intermingling or sequence of these two forms, will apply equally to the prognosis. In other words, the more the signs and course of the disease indicate a simple chronic inflammation, the better is the prognosis; while the more manifest the broncho-pneumonic nature of the disease, and the more marked the signs of caseation and destructive processes, the worse the prospect. And yet the fact, that even in apparently favorable cases a specific inflammation may sooner or later follow a simple one, should warn us against giving a positively favorable prognosis.

The prognosis, however, does not depend solely upon the variety of the disease; other factors are also to be taken in the account, especially the parentage, constitution, conformation of the chest, respiratory power, the previous occurrence of other diseases, and the age. If the patient's ancestors have none of them been consumptives, if the patient himself have passed his youth without scrofula, defective thoracic development, ill health, or dissipation, if he have been hardened to vicissitudes of weather, have always enjoyed good digestion, and have lived to the more mature age of thirty or forty years without symptoms of pulmonary diseases, there is good reason to hope that the affection is mainly of a simple inflammatory character, and that it may either be temporarily arrested or may terminate in a cure by cicatrization.

This hope will be still further confirmed, if the disease run its course with slight fever and loss of flesh, and if the other organs take no share in the morbid process.

On the other hand, the prospect of a speedily fatal termina-

tion will be increased, when the history of the case, and the present condition indicate the rapid development of a broncho-pneumonia.

If the patient be a child, or a person between fifteen and thirty years of age, and of phthisical parentage; if there be a history of repeated pneumonias, pleurisies, or apparently only frequent protracted catarrhs; if the thorax be paralytic, and the digestion feeble; if after measles, typhus, or the puerperal state a febrile disease develops, which can be recognized as an affection of the apex, and which is shown by the physical signs to have extended, and to have undergone degenerative changes within comparatively short intervals; then the chances are in favor of the acute form of phthisis, and the more so in proportion to the elevation of the average temperature above the normal.

As regards the prognostic significance of *individual symptoms*, a very slight *cough* at the outset of the disease generally indicates the simple inflammatory form, but in the later stages it may coexist with complete colliquation. On the other hand, a severe initial cough implies an active implication of the bronchial mucous membrane, and consequently the broncho-pneumonic form. This symptom also influences the prognosis in consequence of the serious harm it produces by exciting vomiting, disturbing sleep, exhausting the physical strength of the patient, and not infrequently by exciting hæmoptysis.

The prognosis is modified also by both the quantity and the quality of the *expectoration*. A very scanty expectoration, if the strength be well maintained, and the general condition be satisfactory, has the same significance as a slight cough under the same circumstances. In the later stages, when there is great prostration, the expectoration may be scanty, notwithstanding the rapid progress of the disease. When the expectoration becomes more abundant, globular, and purulent, while at the same time the fever decreases, and the general condition improves, this symptom indicates a limitation of the necrotic foci by a simple cicatricial inflammation; a process which favors the discharge of the necrosed portions of the tissues and exudation, and is therefore to be regarded as a favorable sign. But if the

quantity of the expectoration increases, while the fever remains the same, or becomes more intense, or assumes a hectic character, it signifies an advance in the destructive process. A more profuse purulent expectoration points to the existence of cavities, and has no other significance, aside from the harm done by its excessive quantity. If the sputa are offensive, and contain products of decomposition, we may apprehend sloughing of the walls of the cavities, hemorrhage, and an extension of the inflammation. An offensive, purulent expectoration, containing black pigment and blood, usually indicates the near approach of death. The detection of elastic fibres is important only as regards the diagnosis.

Hæmoptysis has in itself no prognostic significance, since it may be entirely absent in rapid cases, and may recur very frequently in the protracted ones. But when it is several times repeated in connection with a continuously high fever, it indicates a rapidly extending destructive process. Hemorrhages, which are very profuse or which occur in the later stages of the disease, may prove directly fatal either by producing exhaustion, or by occluding the bronchi and inducing rapid suffocation.

The occurrence of ulcerative processes in the *larynx* is an unfavorable omen, not only in itself but also because it diminishes the prospect of an arrest of the disease. These ulcerations in their earlier stages may sometimes heal, and probably most of us have seen cases in which the course of the disease, when thus complicated, has been protracted beyond all expectation, and in which there has even been at times a slight improvement of the general condition ; but these instances are exceptional.

Pleuritis, when local and dry, has no prognostic significance, but the acute cases, accompanied by considerable effusion, are of a more serious character, because even after the diminution or apparent absorption of the effusion, the course of the disease generally takes a sudden change for the worse.

Mention has already been made of the rarity with which *pneumothorax* is associated with a long continuance of the disease after this event ; in fact, there are no instances on record in which a permanent cure of the phthisis has resulted. The prognosis in this complication is always very bad.

As regards the *organs of circulation*, a continuous increase in the frequency of the pulse is an unfavorable indication, even if it be unattended by an elevation of the temperature. The *quality* has the same significance in this as in other chronic affections. A quiet pulse usually denotes an absence of fever, and is therefore, at least for the time being, a favorable omen.

The most important elements in the prognosis are the *fever* and the *loss of weight*. A persistently high mean temperature is characteristic of the phthisis florida, and is a very serious symptom. Equally ominous are high evening temperatures, or high maxima at any other time, even when the minima are normal, and especially if they are subnormal. A continuous elevation above the normal, with only moderate maxima, is more favorable; still better, normal minima with maxima of only slight elevation. Complete absence of fever is an auspicious sign, at least for the time being.

The influence of the loss of weight upon the prognosis is of much the same character. At the same time it should be remembered that this symptom is not always dependent solely upon an elevation of the temperature, and that inasmuch as the other causes may be of a temporary character it is possible for a restoration of the weight to be effected. But independently of the exciting conditions, a progressive loss of flesh is an unfavorable indication. On the other hand, the maintenance or increase of the patient's weight may be regarded as a very good sign, provided due care is taken that the weighings are made correctly; a precaution which is not always observed in private practice.

The condition of the *digestive organs* has also an important bearing upon the prognosis. This is particularly the case in those forms of the disease which are characterized by early and long-continued dyspepsia, or in which severe attacks of true gastric catarrh repeatedly occur, especially if fever or other causes of loss of flesh are also present.

The rapidity with which the disease usually runs its course after the supervention of obstinate diarrhœa, has already been repeatedly mentioned. Such a diarrhœa probably depends upon ulceration of the intestines, and as this process is arrested only in very exceptional instances, the symptom must be regarded as

foreboding a continuous loss of flesh. When the diarrhœa seems to precede the pulmonary affection, it generally points to the broncho-pneumonic form of phthisis with its caseous destructive processes.

Nor can the occurrence of the *affections* of the *liver*, *spleen*, and *kidneys* be regarded as a favorable indication, notwithstanding the retrograde changes which take place in the pulmonary processes under these circumstances. On the contrary, the increasing anæmia and dropsy destroy the patient's life at least as quickly as the lung disease would otherwise have done by itself.

Treatment.

It is not my purpose in this chapter either to give a historical sketch of the therapeutics of consumption or to gratify the desires of those who are eager for new remedies.

The indications for medical treatment must be derived from the pathology, from a knowledge of the morbid process, and from the accuracy of the diagnosis in the individual case. And to carry out these indications successfully we require a familiarity not merely with pharmacological methods, but also with the general normal and pathological physiology of the body.

In view of the fearful dissemination of consumption throughout all zones, and the terrible percentage of mortality, nearly two-sevenths of all deaths resulting from this disease, it is a most unfortunate circumstance for the promotion of therapeutics that Laënnec should have been misunderstood as holding that all phthisis is dependent upon tubercle, a malignant neoplasm, which is as little amenable to treatment as carcinoma. This misconception has led to a convenient expectancy in treatment; we prescribe only to save appearances, or at most to combat individual symptoms.

The importance of laboring to check the spread of this deadly disease, and to diminish the number of its victims has never seemed to be so urgent as at the present time, when there is a growing demand for more attention to the preservation of health, and when the conviction is gaining ground that this is the main function of medical science. And yet it were idle to imagine,

much more to reasonably expect, that we shall ever be able to extirpate consumption from the human race.

Phthisis is pre-eminently a disease whose occurrence and malignancy are largely due to predisposition, or, in other words, to what is called a constitutional anomaly, of which scrofula is the best example. The first indication, therefore, is to remove this dyscrasia, whether it come in the form of an inherited tendency to the disease, or be acquired from such causes as poverty, bad air, improper food, unhealthy apartments, school duties, factory labor, or a sedentary mode of life. These are all causal conditions which predispose to phthisis, and public hygiene has no duty so urgent as their careful regulation. But for a long time to come public hygiene will content itself with writing and talking, and meanwhile the task must be left to individual effort to do what it can with the means at hand.

In my opinion it would be far better for all concerned if many a marriage were prevented; and it could often be prevented, if the physician, instead of yielding, as is too frequently the case, from an entirely mistaken courtesy or forbearance, were to express his decided opposition. For the sake of avoiding excitement, seed is sown which will bring forth a hundred-fold of sorrow and distress.

In families which exhibit a disposition to phthisis, the newly-born child should, of course, not be nursed by the mother, if she be the testatrix. The next best substitute, as every one will admit, is a good wet-nurse; but a good one cannot always be obtained, and then we are obliged to resort to cow's milk;—but with what distrust and anxiety, since the investigations of Gerlach and Klebs! At all events, the milk should be boiled, and should not be much diluted. The prevalent idea of the concentration of cow's milk as compared with human milk is erroneous, and probably accounts for the better results obtained by the use of condensed milk. It is important, also, that the child should be allowed greater freedom in the movements of its limbs and chest. The traditional swaddling, as practised by nurses and learned by the young mother from them, is reprehensible. Above all, fresh air! This cannot be too strongly insisted upon, and as MacCormack says: "Pure, fresh, untainted air, at all

hours, at all times, and in all places, is the one condition with which nothing should be allowed to interfere." And to this may be added the statement of Clark, that there is no condition, even including heredity, which is more favorable to consumption than a lack of fresh air and exercise. Young children should, therefore, be sent out of doors as much as possible, because the air of the room, however large it may be, cannot compare in freshness with the external air.

At the period of dentition it is well to supplement the milk diet with beef broth. If the child have nursed up to this time, the cow's milk, which is now used on weaning, should be diluted—not with water, but with broth made from good mature beef. If dentition be delayed, or there be a tendency to diarrhœa, some preparation of lime is useful. The warm baths should be exchanged as soon as possible for ablutions of a cooler temperature, and by the second year only cold water should be employed once or twice daily. It is unnecessary for me to enter into all the details of the diet and rearing of children, and the previous remarks were intended only to show what careful attention and detailed advice it is the duty of the family physician to give in these cases. It will often be difficult to secure such management, for our modern life removes the physician more and more from the position of the family medical adviser and guide of the physical education of those around him. And now that medical practice is being converted into a trade—that abortion of progress—this condition of things bids fair to become steadily worse.

If the child of our solicitude do not die in his second or third year from meningeal tuberculosis, and if he escape having many colds or febrile processes, as well as the danger of glandular enlargements from vaccination, he will probably enjoy fair health until the time comes for him to attend school. Compulsory attendance at school is doubtless a necessary provision, but the state and the community are under obligations to see to it that the schools are healthily located, the school hours reasonably short, and a sufficiently long recess given for exercise in the open air. Would that the study of classic antiquity, especially of that model people the Greeks, might at least induce

us to imitate them in their gymnastic exercises and their out-door life, so far as our climate permits!

If the child have not already contracted the epidemic diseases of childhood at home from its older brothers and sisters, the dangers of such contagion will hardly be escaped during the period of school life. These affections—whooping-cough, measles, and also scarlet fever—may either lead to consumption directly, or, by producing scrofula and glandular tuberculosis, may supply foci for subsequent infection.

Hence it is the indispensable duty of the physician to carefully watch the termination of these diseases—especially any respiratory complications that may have occurred—and to avoid pronouncing the child entirely well, or permitting its return to school, until all traces of trouble in the bronchi, lungs, or bronchial glands have disappeared. Attention should be particularly directed to the complete restoration of the nutrition and condition of the blood, when these have been impaired.

We come now to puberty, the period of more rapid growth. At this time, even more than in early life, it is vitally important that in all his occupations, but especially when sitting, the child should be taught to carry the shoulders and upper part of the body in such a position that the chest may expand freely. We can hardly expect that the necessary attention to this point can be given by the teacher, whose classes are overcrowded and whose attention is fully occupied with the subject of the lesson. This duty must be left to family supervision, guided by the advice of the physician.

Exercise which is specially directed to the respiratory organs is very important. Ordinary gymnastics are, of course, beneficial, but they are better adapted to develop certain muscles than to secure proper expansive movements of the chest. In fact, where there is a phthisical conformation, some of the severer exercises—such as those on the cross- and parallel-bars—are apt to overtax the imperfect powers of respiration.

Still more serviceable are out-door exercises with running. The cultivation of the voice in singing is also useful; in my observation trained singers rarely become consumptive.

Special exercises which draw the shoulders backwards and

dilate the upper parts of the chest, unquestionably have considerable influence in altering the form of the thorax and in improving its expansive power. All growing children, or at least those who are predisposed to phthisis or have defective chests, should practise these movements.

For the purpose of hardening the body, cold ablutions or river bathing should be habitually used, care being taken that the skin is thoroughly dried. The meals should consist of plain, well-cooked food, and should be taken at regular hours. A sufficient amount of sleep should be secured by interrupting the sitting or standing position with active bodily movements. Anæmia and incipient chlorosis should be guarded against as far as possible, and the blood kept in good condition as to its quantity and quality, in order to improve the chances of complete absorption or cicatrization, in case inflammatory processes should arise. If they do occur, especially in the respiratory organs, convalescence must be promoted by the free use of restoratives, and the causes by which new inflammations can be excited, or the embers of the old revived, should be avoided for some times afterwards. In such cases it may be advisable to recommend residence in a milder climate during the ensuing winter.

If the *resolution be incomplete* after such an inflammation of the respiratory organs, advantage may be derived from the use of mineral waters containing soda and lime, such as the Lippspringe, Neuenahr or Ems, and from bathing in waters containing chloride of sodium, as at Soden or Reichenhall, situations which are protected, and where one can also enjoy the benefits of fresh air.

For persons who are predisposed to phthisis, the *selection of an occupation* is of no less prophylactic importance.

All occupations which involve a specially sedentary life, or the maintenance of constrained positions, or residence in an impure atmosphere mixed with dust or vapors, are to be avoided; the son of a weaver who has died from consumption should never become a weaver. Occupations are to be preferred which permit more out-door life and exercise.

Inasmuch as scrofula is one of the chief predisposing causes of

phthisis, the greatest attention should be paid to this affection in childhood, and to any traces of it in after life. If we could rid the world of scrofula, we should do a great service in the therapeusis of phthisis. The regulation of such questions of public hygiene as the premature occupation of newly-built houses, the use of cellars for habitable purposes, and the overcrowding of sleeping apartments must be left to the authorities, but still much can be done privately towards the extirpation of scrofula by insisting upon more care in regard to food, fresh air and bathing, and by the appropriate use of cod-liver oil and preparations of iron.

The *extirpation* of accessible enlarged glands, which have failed to yield to such constitutional treatment, has been repeatedly practised, and may be recommended as a justifiable prophylactic measure.

The treatment of actually existing *disease of the apex* will depend in the first place upon the diagnosis, whether the affections be chiefly or solely a chronic pneumonia.

In persons of more vigorous constitution, especially if pain and fever are present, a few wet cups may be applied under the clavicles, or between the scapulæ. So long as any fever or pain continues, the patient should remain in a pure air of uniform temperature, and in winter should keep to his room, which ought to be well ventilated, but in summer he may sit out-of-doors in protected places. Exercise especially of the upper extremities, should be taken only in moderation. The diet must be unirritating; strong beef extracts and broth are better avoided, but meat may be used in moderate quantity by persons who are anæmic. As a rule, wine and beer should be discontinued. In accordance with the views hitherto entertained in regard to the action of soda, the carbonate of soda may be used in simple solution, combined with infusion of digitalis if there be much fever. A moderate amount of exercise of the lungs may be permitted even during this active stage of the disease, with the view of preventing the collapse of the lobules in the affected part which are still permeable, and of hindering, by the free circulation of air in the bronchi, the drying up of substances lying within them.

On the *abatement* of the inflammatory symptoms it has always been a favorite practice to employ local derivation. This mode of treatment seems to be of service, and may be carried out by painting the whole upper part of the chest and the interscapular regions with the tincture of iodine, by inunction with croton oil or tartar-emetic ointment, or by the use of blisters kept open for a long time. The full diet may now be gradually restored, and in order to promote absorption, as well as to effect as complete a development of the cicatricial process as possible, it is desirable to improve the circulation by proper measures adapted to increase the supply of blood, and to strengthen the action of the heart. For the purpose of expanding the aerated parenchyma lying between the foci where we expect the cicatrizations to take place, respiratory exercises may now be used to a greater extent, and advantage may be derived also from inhalations of compressed air. This measure not only benefits the pulmonary circulation, but the increased pressure also assists to dilate the collapsed alveoli and the contracted bronchi, and thus opens a wider surface within the diseased part to the action of the air on ordinary respiration. The greater fulness of the pulse, and the slower breathing after these séances, may be thus explained. Further trials with this method are necessary before it can be decided whether the numerous apparatus which have recently been invented, can replace the bell-apparatus, or are perhaps superior to it.

This training of the respiratory organs in convalescents may also be supplemented by a residence in an elevated protected region during favorable seasons of the year. This measure usually also produces an increase of appetite. But if the bronchial secretion still continue to be excessive, and the respiratory murmur over the affected part still remain at times indistinct, the patient before going to the mountains may first try the soda waters above mentioned, the Lippspringe (also the Inselbad at Paderborn), the Neuenahr, or the Ems, or the alkaline muriatic acidulated waters, such as the Obersalzbrunn, Reinerz, etc.

In view of the great importance of *preventing a relapse*, of ridding the patient of the remains of his arrested disease, and of promoting the expansion of the lung, it is very desirable that he

should avoid a northern climate during the ensuing winter, and this can best be accomplished by southern travel. But the particular latitude or locality which is to be selected will depend upon the condition of the patient and upon the accommodations which the place affords. Cold days and raw winds are to be met with everywhere, and there is no place in which a reasonable prudence is unnecessary, but localities where the patient is obliged to spend the winter within doors are of course to be avoided.

Another desirable prophylactic measure against relapses is that of inuring the skin to the influences of cold. There is considerable uncertainty however in regard to the *modus operandi* by which this result is effected. Runge supposes, and I think correctly, that the explanation is as follows: the vessels of the skin when exposed to cold contract rapidly, under the influence of heat they rapidly expand, and when both of these agents are withdrawn they immediately resume an equilibrium after the reaction. If this explanation be correct, the habitual use of cold, in a degree suited to the susceptibility of the patient, and followed by friction and exercise, must gradually accustom the skin to such adjustments.

It is not improbable that the effect is increased when the water contains considerable salt. The beneficial effects of cold ablutions, shower baths, and sea-bathing in the treatment of pneumonic residua are, in my opinion, to be explained as above.

When, however, we have to deal with the *broncho-pneumonic*, the *specific* form of the disease, or *phthisis proper*, the indications for treatment are different.

If it be admitted that tubercles, and the tuberculous scrofulous inflammatory products accompanying it, undergo no other metamorphosis except the caseous, and that the extension of the process and the healing of already existing destructions can be effected only by the formation of cicatricial tissue around them, the indications for treatment will therefore be, to promote the caseous transformation, to facilitate the removal of caseous matters, and to restore the conditions under which normal connective-tissue forming inflammations can take the place of the tuberculous.

The prospect is, however, far from satisfactory, when we consider the means upon which we can rely.

To the above indications is also to be added another very important one, the reduction of the fever, which is the main cause of the emaciation and wasting of the body.

Again, the treatment will of course vary according to whether we have to deal with a still existing, advancing process or with a period of arrest.

The supposition that the degeneration of the products of tuberculous inflammation can be hastened by the use of certain saline waters is of course a mere hypothesis, and little more can be said of the idea, that by exciting a fluid bronchial secretion the tenacious, crumbling caseous masses may be dissolved and removed by expectoration.

Various other plans of treatment must also be regarded as equally hypothetical; such as the use of whey or grape juice, or the mineral waters above mentioned, which are generally given mixed with milk, or the direct spraying of the respiratory organs with dilute saline solutions or sea-water.

In regard to the latter method, it is still a question whether these inhalations, considering the brief time during which they are employed, really reach the lungs in appreciable amount. Still after such sprayings we often see the expectoration increased considerably, although it must be admitted that its exact nature has never been investigated, and we not infrequently find that the crackling râles and altered respiration detected at the outset of this treatment change after a few weeks to a pure bronchial breathing without râles. Besides this there are still other unpleasant effects from the bold use of this method of employing the natural mineral waters in already existing broncho-pneumonic processes, which have justly brought it into discredit, and which probably arise from the irritating action of the large quantity of carbonic acid, which these waters contain, upon the vascular system.

Carbonate of soda may also be used with milk as a vehicle, or lime-water and milk may be given. The lime-water is also employed by inhalation to modify the bronchial affection.

The action of fat probably also belongs to this category.

When fat is used in an easily digestible form, such as cod-liver oil, good butter, or milk fresh from the cow, its presence in the affected blood-vessels and lymphatics, whose endothelium forms the starting-point for the microscopic miliary tubercle, may so improve the nutrition of these parts as in various ways to limit the formative process of the tuberculous inflammation.

Inhalations of lime and recently of carbolic acid have also been used to arrest the extension of the broncho-pneumonic inflammations. My own experience with carbolic acid inhalations has been so unfavorable that I cannot recommend them; in my cases they proved entirely useless. The great importance of pure air "at all hours and in all places" is indicated here also, as well as in the prophylaxis. If the air in every house is impregnated with organic bodies, which can excite the process of putrefaction, how much more must this be true of the bad air of close, narrow sleeping and sitting rooms! These impurities may not inflict any injury upon a mucous membrane covered with epithelium, but when they lodge upon a denuded or ulcerated surface, or upon masses of exudation their deleterious effects are developed. Great care should be taken, therefore, in broncho-pneumonia, that there should be a constant renewal of air having a uniform moderate temperature.

The extension of the morbid process is probably favored also by the action of other irritants; hence the importance of an unirritating diet. Whether alcohol should be employed in suitable doses to check the destructive processes in this form of phthisis I am not prepared to say; at all events alcohol does not raise the temperature.

By carrying out these indications for treatment, and by facilitating the expectoration of the broken-down products we shall best succeed in promoting the formation of a *limiting zone of simple inflammation*, and in thus arresting the existing process. This object may be furthered also by improving the condition of the blood by agents, which increase the *appetite* and thus improve nutrition, such as *Peruvian bark*. The effects of this remedy, it seems to me, do not depend entirely upon its alkaloids, nor upon the tannic acid contained in it, and hence I gen-

erally use the simple extract of cinchona. *Malt extracts* are to be recommended for the same reason.

The *fever* requires special treatment. This symptom will not entirely disappear until the febrifacient substances are expelled from the body, but still it may be abated by treatment, and thus one of the causes of the extension of the local process may be counteracted.

Digitalis and quinine should be used only for the purpose of temporarily reducing very high temperatures. Neither of them should be given continuously, digitalis particularly, on account of its very injurious action upon the gastric mucous membrane. Our main reliance must therefore be *diet* and *rest of body.* The food should consist principally of milk, the boiled grains, and stewed fruit with a little bread. As the fever abates we may gradually return to eggs, meat, and finally to beef-extracts, wine, and beer.

The patient and his friends, like physicians themselves formerly, are still always in favor of "strengthening" treatment; but it should be borne in mind that we have a definite object in view, viz., the reduction of the fever, which is the chief cause of the physical prostration, and that this reduction promotes the arrest of the local process. When this is accomplished tonic treatment is appropriate. It is, moreover, entirely erroneous to suppose that the weight of the body cannot be maintained upon this restricted diet even in the presence of fever; in fact I have often seen the weight increased.

All these measures are, however, of little or no avail in that form of phthisis in which there is a continuous and rapid destruction of large portions of the pulmonary parenchyma, with a continued high fever; the course of phthisis florida is not to be arrested even for a moment.

In all other forms the symptoms in the earlier part of the disease usually subside for a time, either spontaneously, or as a result of treatment, and in many cases may be completely arrested. In such cases the indications for treatment are much the same as in simple chronic pneumonia, except that the patient is generally more debilitated, more anæmic, and less capable of resisting injurious influences. Ferruginous preparations, and

those which promote appetite, a nutritious diet, pure air, exercise of the lungs, and the hardening of the skin must now be employed judiciously and perseveringly.

In this stage of arrest many persons, who still retain some degree of strength, derive great benefit from the method used by Dr. Brehmer of Görbersdorf, and also at Davos. They generally increase considerably in weight, the cough in some cases disappears entirely for the time being, and many of these patients enjoy a satisfactory degree of health for years. If the nutrition be seriously impaired, the vascular system very irritable, and the larynx be threatened, places of resort must be selected which are less elevated. Those which are free from dust are to be preferred. In some cases the relapse may perhaps be prevented by an expatriation for life or for several years to Egypt, Madeira, or the plateaus of Central America.

If the patient, after an absence of several months, be obliged to return to his home, and again expose himself to the injurious influences of his occupation, a new attack will usually occur in the course of one or two years. Can this be prevented by establishing an issue on the upper part of the arm, as was customary in former times? How much benefit is to be derived from the use of revulsive measures for the purpose of changing the abnormal course of the nutritive processes, it is impossible to say; nor are we able even at the present day to explain how an artificial suppuration can check internal disorganizations, or diminish established morbid discharges, especially suppurations, in internal organs, and yet in all times there has been unbiassed testimony to show that this mode of treatment apparently modifies the course of morbid processes, and particularly those which are of a constitutional character, or are dependent upon a dyscrasia. What we cannot explain may very well be true notwithstanding. As for myself, however, I have never used such derivatives.

The complications and incidental symptoms of phthisis are to be treated in the same way as when they occur independently of this connection.

When the *laryngeal affection* is merely of a catarrhal character it will sometimes disappear under the use of inhalations of lime-water, or solutions of tannin or various salts. Ulcerative

processes may be arrested by the application of mild caustics, and for this purpose a solution of carbolic acid may be tried.

Profuse *suppuration* from cavities may be diminished by the local and internal use of the resins and balsams. Many years ago an English physician recommended the paracentesis and injection of cavities ; this operation has recently been repeatedly performed.[1]

In *pneumothorax* the chief indication is to check the rapidly ensuing pleuritis by applications of ice, and to diminish the shock by means of narcotics, so as to quiet the breathing. With the aid of morphine the patient will be able to lie upon the diseased side, and thus allow the other lung, which is carrying on the whole function of respiration, to expand more easily, because under these circumstances it does not have to raise the weight of the upper part of the body in inspiration. The use of narcotics is also unavoidable at times for the purpose of alleviating the severe cough, which is often dry and incessant. Sleep moreover diminishes the waste of the body.

In *hæmoptysis* the treatment is to be directed mainly to the contraction of the blood-vessels. The favorite methods of carrying out this indication, which I believe to be the correct one, are applications of ice, and the use of lead and ergot, the latter also hypodermically. These measures should be supported by rest of the respiratory organs, which is to be secured by morphine, if necessary, by the maintenance of a comfortable position, and by dry-cupping the lower extremities. The direct employment of agents which coagulate albumen, such as the inhalation of a solution of chloride of iron, has not commended itself to general approval. Such remedies are of course justifiable when there is a constant recurrence of hemorrhages, and the spontaneous contraction of the vessel is impossible.

The *dyspepsia* often baffles all our efforts to control the disease. Neither changing the articles of food or their mode of preparation, nor the administration of small doses of iron with bitters, malt extracts, extract of cinchona, pepsin, and hydrochloric acid seem to do any good. Nor are we much more successful in

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[1] For details consult *Koch*, Berliner klin. Wochenschrift, 1874, No. 16.

the treatment of *diarrhœa*, when this, as is generally the case, depends upon incurable lesions. Still, since we sometimes find considerable ulcerations in cases which have been free from this symptom, we can understand how the usual methods of treatment may at least ward off exhaustion from this cause, even if it do not control the morbid process. Regulation of the food and drink is important, and the various astringents, to which a little opium may always be added, should be tried in succession. The protracted use of *lead* is hazardous. Krameria and hæmatoxylon are sometimes more efficacious than tannic acid itself.

The annoying *perspirations* are sometimes relieved by the usual treatment with mineral acids in astringent decoctions, such as sage tea, or somewhat more frequently by atropine, but very often both fail. With the more vigorous patients ablutions with cold water, to which spirits have been added, may be tried. In Görbersdorf benefit is derived from cold douches and cognac.

ACUTE MILIARY TUBERCULOSIS.

Walter, Ueber die acute Lungentuberkulose. Prager Vierteljahrschrift, Bd. VI., 1855.—*Leudet*, Recherches sur la phthisie aigue chez l'adulte. Thèse. Paris, 1851.—*Rilliet et Barthez*, Traitè clinique des maladies des enfants, Vol. III.—*Wunderlich*, Zur Heilbarkeit der acuten Miliartuberkulose. Archiv der Heilkunde, 1860.—*C. E. E. Hoffmann*, Beiträge zur Lehre von der Tuberkulose. Deutsches Archiv, III., 1867.—*Steffen*, Klinik der Kinderkrankheiten, II. Theil. —*Manz*, Ueber Tuberkulose der Chorioidea. Gräfe's Archiv, 4. u 9. Jahrgang. —*Busch*, Ueber Tuberkulose der Chorioidea. Virchow's Archiv, B. 36.—*Cohnheim*, Ueber Tuberkulose der Chorioidea. Virchow's Archiv, B. 39.—*v. Gräfe* u. *Leber*, Ueber Aderhauttuberkel. Archiv f. Ophthalmologie, B. 14. —*Fox*, Clinical Observations on Acute Tubercle. St. George's Hospital Report, 1870.—*Jürgensen*, Zur Diagnostik der acuten Miliartuberkulose. Berlin. klin. Wochenschrift, 1872, No. 5.—*Hütler*, Zur Diagnostik der acuten Miliartuberkulose. Wiener medic. Presse, 1872, No. 12.—*Buhl*, Bericht über 280 Leichenöffnungen. Zeitschrift für rationelle Medicin, Neue Folge, Bd. VIII.—*Burkart*, Ueber Miliartuberkulose und über das Verhältniss etc. Deutsches Archiv, Bd. XII.

THE earlier writers, who regarded tubercle as the criterion of phthisis, described very acute forms of this disease, which ran their fatal course within a few weeks, and in which the chief lesion found was the presence in both lungs, and frequently in other organs, of a great number of miliary tubercles, which had not yet undergone any further transformation. Nowadays, however, when the miliary tubercles can no longer be considered a necessary accompaniment of consumption, this acute affection, in which the miliary tubercle forms the only, or by far the most important lesion, must be regarded as distinct from phthisis.

That such a distinction should be made is evident, not only from the pathological anatomy of the two diseases, but also from their symptoms, course, prognosis, and therapeusis, all of which

are entirely different in acute miliary tuberculosis from what they are in pulmonary consumption. The only close relationship between them is in respect to their etiology, as has been shown by the pathological experiments which have been made, that is if the very minute tubercles found in these animals were really tubercles. Still even the pathological anatomists have been satisfied of the existence of this relationship. Buhl especially has characterized the miliary tuberculosis as an infective disease produced by an auto-inoculation with caseous matter in the body. In many cases the miliary tuberculosis manifests itself only in the form of a basilar meningitis ; at least the dominant symptoms during life are of a cerebral character. In other instances, however, the secondary eruption of miliary tubercles takes place chiefly or solely in the lungs ; or it may extend further, and involve the serous membranes, the peritoneum and pleura, as well as the liver, spleen, and kidneys. This lesion may be found, moreover, either in persons who already have a chronic or rapid consumption, or in those whose lungs have been unaffected. In the great majority of these cases careful examination will reveal somewhere in the body a caseous mass, from which absorption may be supposed to have occurred. And although occasionally our failure to discover such a caseous mass may leave the causation of the miliary tuberculosis in doubt, yet the results of all the inoculation experiments have been so uniform that one can scarcely avoid the conviction that in man also miliary tuberculosis proceeds from infection, and generally from auto-infection.

In the historical sketch, previously given in my article on pulmonary consumption, it was mentioned that Mangetus was probably the first to recognize true miliary tuberculosis. Then Bayle distinguished phthisis granulosa as a separate form, but his description is very imperfect, and does not correspond to miliary tuberculosis, although it must be admitted that the history of some of his cases (*e.g.*, Obs. 2) shows that they were really of this character. The same may be said of the observations of Laënnec and Louis, who described miliary tuberculosis under the head of acute phthisis.

Probably the first writer to lay adequate stress upon the

special character of this affection was Waller.[1] He divided it into five different varieties, while Leudet[2] was content with three —a typhoid, a catarrhal, and a latent form. The typhoid form has always excited the most interest on the part of physicians, on account of the great difficulty in distinguishing it from typhoid fever, to which it presents a striking resemblance in some cases and at certain stages.

But these writers also, and in fact all except the more recent authorities, regarded this affection as an acute phthisis. The results of inoculation, however, and the constantly accumulating evidence from pathological anatomy, render this view untenable; acute miliary tuberculosis must now be considered not as an acute variety of pulmonary consumption, but as an infective disease, which may occur whenever an opportunity arises for the absorption of caseous matter by the vessels. The existence of such matter in the various forms of pulmonary consumption explains how the latter may become complicated by an acute miliary tuberculosis, and why so many cases of pulmonary consumption terminate in this way.

Again, the fact that the *miliary tubercles* are found in greatest numbers in the *lungs*, and that pulmonary miliary tuberculosis is the chief variety of the affection, is additional evidence that it arises from absorption of infective substances. There are, to be sure, other forms of acute miliary tuberculosis, in which the lungs are unaffected, but these cases form only a very small proportion of the whole number. These exceptions are usually cases of rapidly fatal affection of the pia mater.

In the further description of this tuberculosis I shall, therefore, confine myself to its pulmonary form, and shall speak of the other localizations of the miliary tubercle only as complications.

Etiology.

As has already been mentioned, we know of but one cause for the formation of the miliary tubercle, viz., the absorption of caseous matter. How this produces tubercle requires still further

[1] Prager Vierteljahrshrift, 1845, Bd. VI.

[2] L. c., p. 34.

investigation. Waldenburg supposes, from the fact that the coloring matters used to inoculate animals are to be found in the tubercle nodules, that a transportation of corpuscular elements of some sort takes place. And it is very certain that a transportation must take place when a substance, introduced into the body at one place, produces a formation in another part. The occurrence of a transportation is, however, of less importance than the fact that the substances transported excite tissue-irritation and its results at the point where the secondary formation is produced, and of less importance also than the discovery of the particular structures in which these irritations take place.

In the animal experiments the blood-vessels must probably be regarded as the means of transportation, and the nodules, which are observed, must spring from the small arteries or capillaries. In human tuberculization, however, it is very often the lymphatics which absorb the matter, and produce the gradual diffusion in the body; hence the tuberculization, which is found very frequently along the lymphatics and in the lymphatic glands in the neighborhood of a caseous mass. How a general infection, an acute miliary tuberculosis, is produced under such circumstances it is not easy to say. Probably after a while some of the matter passes into the blood-vessels, but whether previous physical, or also chemical changes, or both together, are necessary for this result is still undetermined. If in a case of so-called tuberculosis of the urinary passages, the miliary complication affects only the meninges of the brain, while the lungs remain intact, it is difficult to explain the mode of transportation.

Most cases in which miliary tubercles are found in different parts of the body present also antecedent lesions in several organs, and it is therefore impossible to decide from which of them the supposed absorption actually took place.

Miliary tuberculosis is found to occur first of all in the lungs, in connection with pre-existing lesions of the apices. It arises also from caseous lymphatic or bronchial glands; hence its connection with measles and scrofulous affections; also from caries of bones, and from caseous affections of the testicles, prostate

or bladder. The same effect may be produced by inspissated abscesses, by the remains of serous inflammations, and by ulcerations in mucous membranes. The latter are probably the efficient cause of the acute miliary tuberculosis, which sometimes follows immediately after typhoid fever. Birch-Hirschfeld [1] and Waller have reported cases of this kind. The latter, in accordance with the standpoint which he occupied at that time, regarded both of these diseases as arising from the same crasis.

Miliary tuberculosis may be produced also by other affections of the most diverse character, provided they give rise to caseous foci. Pulmonary consumption is consequently only one of its exciting causes, but of course one of the most common on account of its great frequency.

Acute miliary tuberculosis occurs also at all ages, but most frequently during the prime of life, the very period when pulmonary consumption reaches its maximum. Children in the first months of life and old men past seventy may be attacked. Sometimes it occurs in vigorous persons, who are apparently in perfect health, at other times in those who are suffering from the anæmia of phthisis.

General Description and Course of the Disease.

The general picture of acute miliary tuberculosis, when its products, the gray miliary tubercles, are formed solely or at least primarily and specially in the lungs, is that of an acute diffuse bronchial catarrh with constitutional symptoms, which resemble sometimes those of exanthematous typhus and sometimes those of typhoid fever. In other cases the predominant symptoms may be of a cerebral character, indicating a local intracranial process, a basilar meningitis, and quite obscuring the symptoms of the respiratory organs. The whole picture of the affection is then changed, and conforms to that of meningeal tuberculosis, which need not here be specially described.

According as this disease attacks persons who are already consumptive, or have suffered from serious morbid processes in

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[1] *Burkart*, Deutsches Archiv, XII.

other organs, or those who appear healthy, that is, whose infective foci have long remained quiet and have not impaired the general health, the attack will manifest itself either as a sudden aggravation of an already existing disease, or as an acute primary affection. Usually it begins with considerable fever, which is often marked by a chill, and is accompanied by prostration, dull headache, perhaps some ringing in the ears, occasionally epistaxis, disturbed sleep and dreams. The symptoms of bronchial catarrh develop immediately. *Increased frequency* in *breathing* is particularly noticeable; at first without special *dyspnœa*, that is, without forcible action of the respiratory muscles. The respirations may vary, according to the age of the patients, between thirty and fifty, and in children between eighty and ninety per minute. Auscultation reveals a general accentuation of the vesicular murmur, whistling and sonorous rhonchi from apex to base, and at a later period crackling râles, which become more and more abundant.

Gradually the respiration becomes also dyspnoic, and distinct attacks occur of more severe oppression and orthopnœa. The contractions of the heart in many cases are very much accelerated from the start, sometimes however not until later, but there is always a constant increase in their number, with a diminution in the filling and tension of the arteries. The rapid pulse—from 120 to 140 and upwards—is disproportioned to the elevation of temperature, for neither is the latter usually very high, nor does it rise at all parallel with the increase of the pulse. The extremities become cool, slight cyanosis makes its appearance in them and in the face, and towards the end the mean temperature falls. Besides these symptoms, which on the part of the nervous system present the form of depression, on the part of the respiratory system the form of exaltation, and on the part of the organs of circulation the form of paralysis, the chief symptom on the part of the digestive organs is anorexia. Sometimes the cavity of the mouth is dry, at other times moist without any special reddening; sometimes there is intestinal catarrh, and the stools may be light-colored; at other times the bowels are constipated, and the abdomen is either normal or slightly swollen. As a rule, a moderate enlargement of the spleen can be detected; the condition

of the urine corresponds to the fever. Not infrequently profuse sweatings occur, and the trunk is covered with sudamina. The prostration increases from day to day, and the amendments are inconsiderable and brief. The form of the fever is variable; generally it is more markedly remittent than in other acute diseases, and not infrequently the maximum occurs in the morning. The rapid loss of strength cannot be ascribed to the fever alone.

The general *course* is rapid and fatal. In all the cases, where the acute miliary tuberculosis supervenes upon phthisis, or follows a typhoid fever, the duration cannot be determined. In the primary uncombined cases it generally averages from two to three weeks from the onset of the fever; still, this period may be exceeded. With the exception of a few temporary amendments of uncertain duration, the course of the disease progresses steadily in all the individual symptoms, and death ensues with collapse.

Morbid Anatomy.

Rokitansky, in the first edition of his handbook, which appeared in 1842, Vol. III., p. 144, says: "The tuberculosis is either *acute* or *chronic*. In the *first case* it attacks, with characteristic symptoms of a typhoid type, both lungs, and frequently also other parenchymatous organs and membranes; the tubercle is the product of a very high degree of the tuberculous dyscrasia, and is deposited either all at once or at repeated rapidly recurring times with periodical exacerbations of the symptoms, and in the form of the most delicate granulations, which have either a transparent vesicular appearance, and are scarcely as large as oat grains, or are gray, crude, and of the size of millet seeds. These tubercles always appear in great numbers; they are discrete, uniformly distributed throughout the pulmonary parenchyma, and only in rare instances do they become aggregated and coalesce in particular places, but even then there is uniformity in their distribution. They all exist in the same stage, viz., that of crudity. At the same time the lung is hyperæmic and œdematous, while its tissue has lost its compactness as a result of emphysema. The hyperæmia may here and there have advanced to pneumonia and hepatization.

In most cases the miliary affection attacks the lungs when the tuberculous disease has already manifested itself in them by the presence of tubercle which has advanced to the stage of fusion and ulceration (vomica), and especially when it has existed for a greater or less length of time as a more or less circumscribed, lingering tuberculosis in the usual situation, that is in the apices of the lungs. The focus of attraction for the acute production of tubercle in the lungs is therefore generally to be found in a pre-existing chronic tuberclosis of the organ."

To this description, it seems to me, nothing need be added, except that in the few cases which present no antecedent phthisical changes at all in the lungs themselves, such lesions, may be found either in the bronchial glands or occasionally in other places, such as the bones, or urinary organs. Leudet found caseous bronchial glands in three-fourths of the cases he examined. The mucous membrane of the trachea and bronchi is sometimes congested, and contains some catarrhal secretion; in the case of the bronchi, however, these lesions may be connected with the older parenchymatous changes.

Outside of the lungs the miliary tubercles are also found, usually in their minutest form, in the spleen, liver, kidneys, upon the serous membranes, and in the choroid, where they were first demonstrated in the living subject by Manz, and in the cadaver by Cohnheim. These numerous tubercle-eruptions are frequently surrounded by signs of hyperæmia, but they show no tendency to further changes, except that some of them, especially those in the lungs, become more opaque, and here and there slightly yellowish. At all events the tubercle-eruption cannot be regarded as the cause of death, because in all the organs in which it occurs there is still left a sufficient amount of parenchyma capable of fulfilling its functions.

The signs of rapid decomposition in the cadaver, the hypostases, and the diminished coagulability of the blood ally this affection to the acute infective diseases; as does also the acute enlargement of the spleen, which is generally found even in those cases which are unattended by miliary tuberculosis of this organ.

Analysis of Individual Symptoms.

The symptoms of the *respiratory organs* are constant. The cough is never absent, and is specially severe, at least at the outset of the disease, although later it diminishes somewhat. It is not characteristic, and often consists of very frequent, short, dry concussions, rather than of longer paroxysms followed by expectoration, as in catarrh. This cough must be ascribed to the hyperæmia of the bronchial mucous membrane, because it is improbable that the tubercle-eruption in the parenchyma itself should be able to produce it. The abatement of the cough in the latter part of the disease may proceed chiefly from the diminution of irritability which we must infer takes place in the medulla oblongata as well as in the cerebrum.

In some cases there is no *expectoration* at all, in others it consists of simple, colorless, rather viscid mucus, sometimes mixed with streaks of blood. Larger hemorrhages are not due to the acute miliary tuberculosis, but to the phthisis, which may have preceded the latter. Such attacks are, however, very rare.

The most striking symptom on the part of the respiratory organs is the *alteration* in the *breathing.* It is only exceptionally that a *considerable increase* in the frequency of the respirations has not been noted. Usually in adults the respirations run up to from fifty to sixty per minute, but at the outset this acceleration is not attended by the increased exertion of the respiratory muscles, which is characteristic of true dyspnœa. And as the latter is always the immediate expression of an accumulation of carbonic acid in the blood (Traube), the form of accelerated breathing alluded to must be produced by irritation of centripetal nerves, probably from the action of the numerous tubercle-eruptions upon the peripheral filaments of the vagus in the lungs. A certain degree of irritation applied to the central end of the divided vagus produces, as is well known, an increase in the rapidity of the contractions of the diaphragm (Traube). But besides this striking acceleration of the breathing there occurs also, as the case progresses, more and more marked *dyspnœa*, sometimes in the form of severe paroxysms, which prevent the patient from maintaining the horizontal position. The

patient usually also complains of a distressing sensation of pressure or constriction at the lower part of the sternum.

This dyspnœa is evidently produced, or, in other words, the exchange of gases in the alveoli is diminished, partly by the accumulation of secretion in the bronchi and by the inflammatory or œdematous infiltration of the parenchyma of the lungs, and probably also partly by stasis in the pulmonary circulation resulting from the depressed force of the heart.

The *physical signs*, independently of those connected with the preceding phthisical changes that may have taken place in the upper lobes, are merely those of the diffuse bronchial catarrh.

In particular, there is seldom noticed any abnormity in the *percussion note;* at most only some impairment of the tone at various parts of the chest as compared with the corresponding situation on the other side, or a slight tympanitic quality, changes which are not constant, but alternate on repeated examination.

Auscultation reveals the different signs of catarrh: accentuated inspiration with audible expiration, whistling sonorous rhonchi over the whole lung, and at a later period different kinds of crackling râles, generally those proceeding from bubbles of small or medium size, heard first in the dependent parts, and from there extending to the anterior and upper regions. Jürgensen has heard a soft friction murmur, which from the fact that the sound was quite diffuse and unaccompanied by pain, he ascribes to an unevenness of the pleura pulmonalis produced by numerous prominent nodules. Further observations are necessary to show whether this is a frequent sign, or is to be regarded as exceptional. Burkart found the murmur rough where the tubercles were obsolete.

On the part of the *organs of circulation* there is a striking frequency and feebleness of the cardiac contractions. The pulse in adults is not infrequently as high as 120 during the morning, and during the latter part of the disease from 130 to 150. At the same time it is soft and small. The livid cyanotic color of the nails, and also of the nose and lips, is evidently the result of the obstruction to both the respiration and the circulation.

In other respects the skin is abnormally pale, and the face is

rarely flushed, notwithstanding the skin feels warm or even hot. The lips become dry, and even fuligo dentium, with dryness of the tongue, may supervene, for there is always an active *fever*. The fever has no definite type, and varies considerably in the same patient. Sometimes the disease begins with a continuous fever of moderate severity, then passes into the steep curves of hectic, and finally sinks gradually into the temperatures of collapse.

At another time the hectic form prevails from the start, and very high maxima alternate with low minima, so that daily variations of from four and a half to five and a half degrees may occur; or the fever begins as in typhus [abdominalis], rising rapidly into a high continuous type, and in the third week changes into an irregular remittent. Repeated perspirations usually occur, sometimes daily, especially in the strongly remittent forms.

At all events it is the diversity and irregularity of the curves in acute miliary tuberculosis which distinguish it from the other febrile diseases, with which it is apt to be confounded. In an individual case, however, the fever curve is insufficient by itself to enable us to recognize the disease, but taken in connection with the other symptoms it is always a valuable indication. Moreover, the inverse type, with high morning and lower evening temperatures, has been found in miliary tuberculosis more frequently than in other febrile diseases.

The symptoms on the part of the *nervous system* present the greatest diversity, according as the meninges are involved in the acute miliary tuberculosis or not. In the former case, the meningeal symptoms are so predominant that the characteristic respiratory and circulatory disturbances, previously mentioned, are considerably modified and no longer recognizable. These cases, on account of their essentially meningeal character, will not be described here. In other instances, where the meninges of the brain are entirely unaffected by the tuberculous process, or are involved only to a very slight extent, the nervous symptoms are allied chiefly to those of the typhoid diseases. Mental dulness, vertigo, and occasionally some frontal headache are not infrequently observed here, as well as in the meningeal form.

To these symptoms may be added apathy, generally slight delirium, and great muscular prostration. If the patient be not disturbed by attacks of severe dyspnœa, he lies soporose, and finally becomes comatose.

Much less constantly functional derangements of the *digestive organs* are noticed. The condition of the buccal cavity varies considerably; sometimes it is dry, and the gums and tongue are covered with black incrustations from the drying of the blood proceeding from the slight hemorrhages, which are apt to occur; at other times the mucous membrane is pale, and the tongue remains moist. The abdomen is generally of normal volume; it may, however, be swollen, but there is no sensitiveness at particular points. Its skin is sometimes covered with sudamina, but whether true roseola occurs is undetermined. Waller mentions it, but others, including myself, have never seen it.

The *spleen* is almost always moderately enlarged, but neither is the increase during the first weeks important, nor still less can any decrease be detected during the later ones. It is only when there is a very considerable deposit of miliary tubercles in the organ that it increases to the size of the typhus spleen, and becomes tender on pressure.

The *appetite* is always deranged in proportion to the fever; sometimes there is complete anorexia, while at other times the patient is able to take more or less nourishment.

The character of the *discharges from the bowels* varies according to the presence or absence of intestinal catarrh. If this be absent there may be constipation; otherwise diarrhœa may occur to a greater or less extent, but without anything characteristic in the discharges.

The *urine* also presents no special changes beyond those due to the fever. It is somewhat darker than usual, rarely throws down any sediment, sometimes contains traces of albumen, and in the higher grades of cerebral disturbances it is discharged involuntarily.

Complications.

The *complications* of acute miliary tuberculosis belong in the main to phthisis. It has already been mentioned that at various

stages of pulmonary consumption a miliary tuberculosis may occur, which converts the hitherto mild slow course into an acute rapidly fatal one, and that in many cases lesions of older date are found in the upper lobes. Here the miliary tuberculosis is, to all appearance, a complication of the phthisis, and the cause of its rapidly fatal termination. In the same way a generally diffused miliary tuberculosis may supervene also upon a number of other diseases, which produce caseous matter, and these may also be regarded as complications; but in the majority of cases which have been observed, it was impossible during life to discover the existence of infective foci.

The relation between miliary tuberculosis and typhoid fever must be considered in this connection. Reported cases seem to show that these two affections sometimes occur together, the tuberculosis developing itself either during the height of the fever or immediately after its termination. In these cases the infection is supposed to arise from the matter of the typhoid ulcerations of the intestine (Birch-Hirschfeld). This complication is, however, rare, and the recognition of the miliary tuberculosis in such cases is extremely difficult (Waller, Burkart).

Diagnosis.

It is evident from the preceding description that there is no single symptom nor group of symptoms sufficiently distinctive to make the existence of acute miliary tuberculosis a necessary inference during life. The certainty of the diagnosis can only be approximative. Especially in cases where the patient is not seen until late in the disease, and where no accurate history or information in regard to its previous course can be obtained, it is almost impossible to avoid mistaking it for typhoid fever. Indeed it may happen that the diagnosis, which seemed to be justified by the symptoms during life, proves to be false at the autopsy; for to diagnosticate means not to guess, but to know. But knowledge must be based upon reasons, and with our present information these reasons may be untrustworthy.

The following may be mentioned as general differential indications. First, the age of the patient; for at all events typhoid

fever does not occur in the first years of life nor in advanced age. Moreover, an early occurrence of anæmia and cyanosis with active fever; a considerable acceleration of the breathing without dyspnœa; a frequent soft pulse without corresponding elevation of temperature; an irregular course of the fever with the inverse type; an absence of roseola and of intestinal symptoms, such as cæcal pain, characteristic diarrhœa and meteorism; a moderate enlargement of the spleen; and early prostration with a steady increase of the same; these indications are all in favor of miliary tuberculosis. The probability is also strengthened by evidence of already existing disease in the pulmonary apices, or of foci elsewhere from which infection can be inferred.

Let us now compare the acute miliary tuberculosis with typhoid fever and acute bronchial catarrh, the diseases with which it is most apt to be confounded.

At the outset of typhoid fever the bronchial catarrh is very rarely so general and intense as in this disease, while the headache and insomnia are generally much more marked, and the rapidly increasing fever is accompanied by very little or no cough. But even if there is considerable bronchial catarrh from the start, as is especially the case in exanthematous typhus, the striking frequency of respiration, which characterizes the tuberculosis, is not noticed. In these typhous diseases, moreover, the enlargement of the spleen is usually greater from the beginning, and the growth of the tumor more noticeable. If an eruption of roseola make its appearance on the epigastrium and hypochondria, either as a general exanthem on the fifth day, or in a scanty form between the seventh and eleventh days, miliary tuberculosis may be excluded, since there is no satisfactory evidence that such an eruption occurs in this affection. Marked intestinal symptoms, epistaxes, and quickly ensuing dryness with redness of the buccal surface point in the same direction. At the beginning of the disease the fever, as far as the course of the temperature is concerned, is an uncertain guide; but in tuberculosis the pulse is more frequent, and soon becomes softer, while the hot skin is paler and is apt to be cyanotic. The cerebral symptoms are also usually different: in tuberculosis the active

delirium, loud talking, and the other more striking indications of excitement are wanting, and this is usually the case also with the ringing in the ears and deafness; on the other hand, the muscular weakness and apathy are generally more considerable from the outset. The discovery of miliary tubercles in the choroid may be regarded as a very positive indication of the tuberculosis. This lesion produces no subjective symptoms, and must be sought for by direct examination.

In the later stages, in which the typhus eruption may have disappeared, an exanthematous typhus may be distinguished by the course of the fever. In this disease the fever is uniformly high up to the end of the second week, and then usually falls suddenly with an outbreak of perspiration and rapid subsidence of all the symptoms. In typhoid fever, also, the course of the fever is very important; there is a regularity in the evening maxima and morning minima, although during the third week the fever becomes more remittent, which is not noticed in miliary tuberculosis. In the latter affection, morever, the perspirations are more frequent, more copious, and without influence upon the continuance of the fever; while in typhoid fever this symptom is less marked, and occurs at definite times, viz., at the end of the weekly periods. An inverse type of fever is wholly in favor of miliary tuberculosis. If diarrhœa be present, a symptom which is still more apt to lead to mistaking the disease for typhoid fever, the evacuations lack the characteristic flaky sediment containing abundant triple phosphates. The occurrence of intestinal hemorrhage at the beginning of the third week makes the diagnosis of typhoid fever certain. In the later stages of typhoid fever, when there is considerable bronchial catarrh, the examination of the chest will usually reveal a more extensive dulness over the posterior inferior regions, resulting from the atelectases and hypostases, which are almost always present in this disease, but are rarely met with in miliary tuberculosis, probably on account of the more active dyspnœa.

From *acute bronchial catarrh* or *bronchitis*, the miliary tuberculosis is to be distinguished by the rapid loss of strength, the frequency of the breathing without simultaneous dyspnœa, the longer duration of the dry stage of the bronchial affection,

the early occurrence of cerebral symptoms, and the enlargement of the spleen.

Miliary tuberculosis may be inferred, also, in all cases in which a diffused friction murmur can be heard without any other symptoms of pleuritis being noticed.

Duration, Terminations, Prognosis, Treatment.

The duration of the disease varies within tolerably wide limits; yet Leudet's statement, that it averages only thirty odd days, would still be true at the present time. Cases undoubtedly occur which are fatal within two or three weeks; but it cannot be denied that the disease may also last as long as five or more weeks. At all events, the most rapidly fatal cases are those in which the miliary tuberculosis is most wide spread. It may naturally be supposed, also, that when the infective process advances by starts, or meets with special obstacles, the course of the disease may be more prolonged.

It is the rapid cases which are most apt to assume the typhoid form; that is, to present cerebral symptoms, which are probably dependent upon the fever, and not upon a true meningitis. In the more protracted cases the disease sometimes manifests itself as an acute bronchial catarrh, with occasional amendments, which are, however, only very temporary; or, in other words, the respiratory symptoms are predominant throughout, and constantly increase in severity. At other times the disease assumes the form of an intense febrile affection, which, without presenting any conspicuous character, and accompanied by gastric and broncho-catarrhal symptoms, runs a latent course, and is not suspected until the increasing prostration and ineffectiveness of all treatment call attention to its serious nature.

The result in all varieties of the disease is almost universally fatal; sometimes in the form of progressive exhaustion, sometimes from increasing dyspnœa, and sometimes with coma. Several cases have been reported in which very numerous miliary tubercles were found in the lungs at the autopsy, presenting all the characters of rigidity, and even a chalky hardness; and hence it has been proclaimed that even miliary tuberculosis is

curable. When an acute miliary tuberculosis is diagnosticated from the symptoms during life, and the patient ultimately recovers, it will naturally be objected that a mistake has been made ; a mistake, of course, to which any one is liable. In the present condition of our knowledge of pathological anatomy it is still necessary to inquire first whether these hard, so called obsolete, chalky nodules, however numerous they may be in the lungs or in any other part, are really miliary tubercles. Hitherto the statements in regard to these curative processes have been based upon the discovery of such hard calcareous nodules ; but these may have an entirely different origin, and it is more probable that one of the characteristic peculiarities of the miliary tubercle is that it terminates only in decay.

Notwithstanding the cases reported by Waller, Wunderlich, Lick, Burkart, and others, the question of the curability of acute miliary tuberculosis must, from a clinical point of view, probably still remain an open one ; consequently when the diagnosis of "acute miliary tuberculosis" is made, the prognosis is necessarily bad ; and if, notwithstanding this prognosis, the patient recovers, the correctness of the diagnosis may be doubted. At least, in the two cases of recovery which I have seen, where this diagnosis was made, I prefer to suppose that a blunder was committed.

A *therapeusis*, in the true meaning of the word, does not exist, unless we credit the idea that the iodide of potassium can check the development of the tubercles, or that some other remedy can effect the absorption or obsolescence of those which are developed.

Since the fever has evidently a considerable share in all the dangerous symptoms, the attempt should be made to effect its reduction. Moreover, since the steadily increasing exhaustion of the heart is one of the chief causes of the early death, the use of cardiac stimulants is indicated ; and probably none are more effectual than moderate and properly repeated doses of a stronger wine.

CHRONIC AND ACUTE TUBERCULOSIS.

RINDFLEISCH.

CHRONIC AND ACUTE TUBERCULOSIS.

I. TUBERCULOSIS AND SCROFULA.

History.

ANY one who wishes to give a special account of tuberculosis of the lungs must necessarily commence by stating his views on the subject of tubercle in general. For so much has been written, and so many different opinions prevail concerning tubercle, that unless an author first states to what conditions he applies this word he cannot be understood. This is particularly necessary in my case, as it is my intention once more to enlarge the domain of tuberculosis of the lungs from the somewhat narrow limits into which it has been circumscribed. And I shall endeavor also to give support from pathological anatomy to those who have always from clinical observation believed that pulmonary tuberculosis is essentially a tubercular disease.

It is well known that there was a time when pathological anatomy pointed with pride to its knowledge of phthisis tuberculosa. Laënnec's theses concerning the gray granulation and its change into yellow tubercle spread, after long controversy, a welcome light over the nature of the entire process. The manifest resemblance of the lesions in different organs, especially in the lungs, kidneys, and mucous membranes, were explained by the axiom that there was only one phthisis—a phthisis tuberculosa. The recognition of Laënnec's views by Rokitansky in his handbook, and their adoption by most clinical teachers, soon planted them widely in Germany. But the opposition to this theory was never entirely silenced, and since 1844 has been more vigorous every year. The weak point of Laënnec's teaching was recognized and assailed. Not all the cheesy masses which we find in phthisical lungs are formed from gray granulations; most of them, even all of them, are simple products of inflammation—this was the reproach constantly urged against

Laënnec's doctrine. Virchow especially, avoiding the errors of Andral and Reinhardt, founded the new doctrine to supplant Laënnec's teaching. He taught that only the miliary tubercles were to be called tubercle, and that no process was to be called tubercular unless the gray miliary granulations were found. Cheesy conditions could be formed from thickened pus and other cellular new-growths just as well as from miliary tubercles. It should be the office of pathological anatomy to separate the cheesy products of inflammation from cheesy degeneration of miliary tubercles. In this way phthisis of the genito-urinary apparatus was held up as the prototype of a true phthisis tuberculosa, the tuberculous nature of phthisis of the intestines and larynx was considered problematical, and pulmonary phthisis was almost entirely removed from the domain of tubercle, and ascribed altogether to a cheesy broncho-pneumonia. So in spite of the agreement of the clinical symptoms and anatomical lesions, in spite of the frequent combination of the different varieties in the same individual, in spite of the similar etiological conditions, the chain was broken which had been formed of the tuberculous phthisis of different organs, and the most important link—pulmonary phthisis—was altogether taken away.

Practical medicine, however, for a long time regarded these novelties with distrust. Felix Niemeyer was the only clinical teacher who boldly adopted the new doctrine. The new investigations and theories were carried on principally by experimental pathologists. The old question of the infectious character of tuberculosis was revived by Villemin. Every one began to inoculate rabbits and guinea-pigs with fresh miliary tubercles, with cheesy matters, and with sputa from phthisical patients. They arrived at the astonishing result, that "in certain animals" these inoculations were followed by positive results, by the development of an acute, even fatal, miliary tuberculosis. Long before this time Buhl had promulgated the idea that miliary tuberculosis was a "resorption disease." Virchow had called attention to the fact that in almost all cases of acute, disseminated miliary tuberculosis cheesy forms could be found somewhere in the body, usually a cheesy lymphatic gland. In the experimental inoculations, therefore, it was interesting

to note that in some cases the miliary tubercles were found in greatest numbers around a cheesy focus, as if the latter were the point of inoculation and resorption. In this way the miliary tubercle lost somewhat its character as a primary lesion, and seemed rather to be a result of resorption and dependent on certain anterior conditions. Then Waldenburg demonstrated that "in certain animals" the manner and matter of the inoculation are of no consequence. After him came Cohnheim and Fränkel, who proved that "in certain animals" it was not necessary to inoculate at all, and that the formation of a focus of suppurative inflammation in a rabbit or guinea-pig was sufficient to render the animal tuberculous. From all these experiments, it became evident that the species of animal employed for experiment was a matter of importance. In rabbits and guinea-pigs any focus of purulent inflammation is very apt to pass into the cheesy condition. It seems very natural, therefore, to suppose that the predisposition of these animals for tuberculosis is a consequence of their disposition to cheesy inflammation, and that such animals become inoculated from their own inflammatory products.

Scrofula.

Thus far reached the experiments and observations on animals. Their principal object was to demonstrate the infectious nature of tuberculosis, and the existence of a tuberculous virus, which, like syphilis, could be transmitted from one person to another. Such an idea I am far from rejecting. On the contrary, I shall have frequent occasion to refer to it when I speak of the histology of tubercle. But for the present I think it more important to employ these experimental results in another way, and to call attention to the remarkable similarity between the predisposition of "certain animals" for tuberculosis, and the almost exclusive occurrence of tuberculosis in a special group of persons—the scrofulous.

It is the characteristic nature of the constitutional disease called "scrofula" that all the inflammatory processes which occur in certain individuals run a peculiar course. Whilst in a normal individual an inflammatory infiltration of any portion of

connective tissue either resolves or suppurates in a moderate space of time, in scrofulous persons the same inflammation shows a well-marked tendency to be protracted. The infiltration disappears very gradually, or it remains stationary, and undergoes regressive metamorphoses of a cheesy character. The question as to the reason of this peculiar behavior of the products of inflammation has busied the physicians of every age. The more ancient authors assumed the existence of a morbid material in the blood, which could stop up the pores of the tissues when an exudation took place. Laënnec believed that every cheesy exudation was formed from transparent, gray granulations, and thus opened the way for the idea of a solid new growth. Andral laid stress on the drying and thickening of a product previously purulent. Rokitansky considers the yellow tubercle to be an exudation "of altered protein matter." Virchow first called attention to the predominant cellular character of the scrofulous exudation; to its hyperplastic nature, and to the low vitality of the cells which compose it. For my own part I would add that fresh scrofulous exudations contain relatively *large* cells with glistening protoplasm, and a nucleus in the act of segmenting, or containing a double nucleus. I have received the impression that the emigrated white blood globules, which in normal individuals pass from the blood-vessels of the inflamed tract to some adjoining surface, or to the lymphatics and lymphatic glands, or become collected into abscesses, in scrofulous persons have a tendency to grow larger on their way through the connective tissue. They swell up by the intussusception of albuminous substances, and in this very swelling die and slowly degenerate.

The consequences of this peculiar anomaly of vegetation are felt in all the inflammations of scrofulous persons; less in the superficial catarrhs of the skin and mucous membranes than in the deeper parenchymatous inflammations of the glands and viscera.

In scrofulous catarrh attention has long been called to the abundance of cells, and the thick, quickly drying character of the secretions. I not only fully endorse this assertion, but I also find a great difference in the degree of cellular infiltration of the submucous connective tissue, according to the scrofulous

or non-scrofulous nature of the catarrh. In the former case the exudation bodies lie so thickly together that they form a layer touching the epithelium, and there is an infiltration, with round cells extending deeply into the submucous tissue. Until these cells have entirely disappeared the mucous membrane is not restored to a perfectly normal condition. Many of these cells wander gradually to the free surface, and are cast off, others pass into the commencements of the lymphatic vessels, while others undergo a granular-fatty degeneration. Their detritus is partly mingled with the lymph which flows from the inflamed tract into the neighboring lymphatic glands, and partly forms an element in the secretion, in which fine granules, possessed of molecular movement, are constantly found.

Is the formation and transportation of the tubercular poison effected by the formation and transportation of this detritus? It almost seems so; but we must for the present be satisfied with indicating its possibility. Although, as we have seen, many ways are open for the elimination of the exudation from the mucous membrane, yet the infiltrated condition lasts for a remarkably long time. Especially persistent is the sub-epithelial layer of exudation already mentioned, and here the inflammatory new growth takes on a more permanent character. This fact is of special importance when we remember that the sub-epithelial connective tissue is the seat of the first miliary tubercles that are formed in cases of tuberculous ulcers. I believe that the reason why the scrofulous catarrh of children is not always followed by ulceration and tuberculosis is, that in young persons there is a marked tendency to active congestion, and that by this active congestion a supply of nutritive material is afforded which keeps the lymphatics open, and leads to the formation of healthy granulation tissue. This same reaction of the vital forces by which a healthy inflammation supplants a scrofulous, although more common in children, occurs also in adults, and it is only in this way that a real cure can be effected.

The deeper parenchymatous inflammations which take on the scrofulous character are much more characteristic. They begin always in the connective tissue of the glands and of other compact organs. Virchow has attempted to distinguish ordinary

from scrofulous inflammation, by ascribing a more hyperplastic character to the latter. In my opinion, however, we can obtain a more correct idea of the real nature of scrofulous inflammation, if while we recognize its inflammatory character we compare its peculiar processes with those of simple inflammation. Thus we will notice at once the tendency of scrofulous inflammation to permanent infiltration of the affected tissues. In simple inflammation the infiltration is a temporary condition which terminates in suppuration, in organization, or in resolution. In scrofulous inflammation the only termination is a cellular infiltration of connective tissues. By this infiltration the connective tissue is converted into a hard, dense, grayish, half-translucent mass, which constitutes the acme of the process. In such a dense infiltrated tissue the blood-vessels become occluded and then necrotic processes ensue. At least there is as yet no proof that scrofulous infiltration is capable of any changes except degenerative ones. This degeneration begins as a cheesy transformation, first of the centre, then of the entire infiltration. After the cheesy degeneration may follow calcification or softening.

The simplest examples of such a scrofulous cheesy inflammation are seen in the testicle and brain. I have shown elsewhere that both the tubercular testis and some of the solitary tubercular tumors of the brain are not produced by an aggregation of miliary tubercules but by this species of inflammation. It is well known that the same kind of inflammation plays an important part in pulmonary phthisis. But in the lungs the process is complicated by the accumulation of inflammatory products within the air-cells. I possess, however, one preparation of a child's lung, in which the anterior portion of the middle lobe was converted into a dense, cheesy white mass, looking like a solitary tubercular nodule in the brain, and in which there was no exudation within the air-cells, nothing but the thickening of their walls. The mass was composed of one large and numerous small nodules, which had all become aggregated together. The origin of the small nodules in the peribronchial connective tissue, and their growth, could be followed out step by step.

The alveolar walls are swollen by the cellular infiltration, the alveolar cavities become smaller and smaller until they dis-

appear, and in place of the lung tissue nothing is seen but an homogeneous mass composed of round cells and connective tissue. There is here, therefore, a growth of connective-tissue cells entirely analogous to that produced by interstitial inflammation of the liver and kidney. But while in the liver and kidney we can demonstrate by injections that the new tissue is very vascular, in this specimen of lung no injection will penetrate into the homogeneous nodules. This shows that the infiltration does not lead to new growth and organization, but to a permanent thickening of the infiltrated parenchyma, which is necessarily followed by cheesy degeneration.

How Scrofulous Persons become Tuberculous.

The final degeneration of the scrofulous infiltration is effected by a chemical metamorphosis, which converts it into fat globules, albuminous granules, and a quantity of soluble substances which cannot be seen. All these substances, however, must necessarily be absorbed.

Now, when we consider that scrofulous persons are especially predisposed to tuberculosis; that tuberculosis hardly ever occurs except in scrofulous persons; that tuberculous phthisis is only a combination of scrofulous inflammation and tubercles; and that in scrofulous persons an inflammation brings with it the risk of tuberculosis, we can hardly fail to see that in certain men, as in certain animals, inflammation runs a peculiar course. The cheesy infiltrations and suppurations of mucous membranes elaborate a poison which, when absorbed, produces tubercles. This constitutes the real relationship between scrofula and tuberculosis. The tubercular poison in most cases is thus manufactured by the patient himself, and it has not yet been demonstrated that this poison can be transmitted to perfectly healthy persons, so that the disease can hardly be considered purely infectious. On the other hand, pulmonary phthisis is almost always a general disease. There is first scrofula and then a cachexia from the absorption of scrofulous products. The intensity of this cachexia is only partially revealed by the formation of miliary tubercles.

I will try to render my meaning clearer by some examples. If a scrofulous child is struck by some accident on the elbow, the joint may become inflamed. Such an arthritis, accompanied with pain and congestion, may last for several months; then takes on a fungous character, and the cavity of the joint becomes filled with pus. The cells of this purulent secretion degenerate, their detritus remains in contact with the diseased synovial membrane, is absorbed, and there results from its absorption local and general miliary tuberculosis. If in some other child, from bad food, a catarrhal inflammation of the small intestine is excited, the adenoid tissue of the mucous membrane becomes infiltrated with cells; these cells break down and are absorbed by the lymphatic vessels. Then there are produced miliary tubercles along the course of the lymphatics up to the mesenteric glands, and even beyond them, so that there is general tuberculosis. In the same way a scrofulous ophthalmia, an impetigo, or a scrofulous ozæna may lead to tuberculosis of the cervical lymphatic glands, and then of the entire body. A catarrh of the larynx, due to cold, may give rise to a scrofulous infiltration of the vocal cords or the folds of the glottis. The cells of this infiltration break down, and their *débris* excite, on account of the unfavorable conditions for absorption, an eruption of miliary tubercles in the vocal cords, followed by tubercular ulceration. In this situation, however, the sequence of morbid changes may be somewhat different. In mucous membranes which are arranged in folds, or which are thrown into folds by their natural movements, or in which opposing surfaces are rubbed together, the tubercles are first found at points of contact. For it is at these points that the degenerated and poisonous products of inflammation are rubbed into the mucous membrane.

Disease of the Lymphatic Glands.

The lymphatic glands may become enlarged and cheesy, and in this condition they have always been regarded as one of the pathognomonic products of scrofula. Virchow agrees with the older authors on this point in a measure, for he considers the change to be an inflammatory one, with hyperplasia of cells,

but without the presence of tubercles. I regret that on this point I cannot agree with my honored master. I have convinced myself by repeated observations that Schüppel's assertion, "that scrofulous glands are always tubercular glands," is correct. Schüppel does not rely for proof alone on the presence of giant cells in the grayish parenchyma; still less do I. The grayish parenchyma of a scrofulous lymphatic gland, which has not yet become cheesy, is studded not merely with a few giant cells, but with a considerable number of veritable tubercles. These tubercles may be called, after Virchow, submiliary nodules. They nearly all contain at their centres a giant cell; around this, adenoid tissue with large cells; and around this, an infiltration of small cells—apparently the parenchyma of the lymphatic gland in a condition of inflammatory hyperplasia. The tubercles are often aggregated in groups (Virchow's conglomerate tubercles), or they may be more uniformly disseminated. But they appear everywhere as the anatomical centres of the inflammatory and hyperplastic changes, so that it seems as if these changes were secondary to the formation of the tubercles. There can, at least, be no good reason for doubting the veritableness of the tubercles described by Schüppel as existing in the lymphatic glands. On the contrary, in tuberculosis, as in the case of malignant tumors, the lymphatic glands are the situation in which the new growth is developed in the clearest and best defined manner. Only in tuberculosis the demonstration requires more care.

If I am right, therefore, in considering the so-called scrofulous glands to be really tubercular, then in scrofula and tuberculosis there exists the same sequence as in the invasion of malignant tumors. But we must admit that the primary catarrh or inflammation is not tubercular, except in the case of tubercular ulcers, and the process does not acquire a specific character until its secondary development in the lymphatic glands.

In tuberculosis, as in the case of malignant tumors, the swelling and obstruction of the lymphatics may cause the disease to remain for a time localized in the glands. We all know for how many years a scrofulous gland may remain as the only trace of

youthful scrofula. It is eminently proper in such cases to adopt Hueter's plan and extirpate such a gland in order to prevent the gradual infection of the whole body with tubercles from it. By primary tuberculosis, then, I mean local affections of the different organs, composed either of both scrofulous inflammation and tubercles, or of the first alone. By secondary tuberculosis I mean the tuberculosis of the lymphatic glands, and by tertiary the general tuberculosis which may involve all parts of the body.

The Anatomy of Tubercles.

In considering the anatomy of tubercle I must make the preliminary remark that the time is past when one can be satisfied with proclaiming "miliary" tubercles as the specific product of tuberculosis. Virchow has already demonstrated that tubercles of the size of a millet seed are infrequent, while those as large as a poppy seed are much more common, and that the size of the nodules is not at all a safe criterion of tubercle. He shows that the smallest nodules visible with the naked eye are made up of yet smaller nodules, the submiliary tubercles. These measure from one-fifth to one-seventh of a millimetre in diameter, so that from forty to fifty of them must be conglomerated to make a nodule as large as a poppy seed. There are no definite limits to the size which may be attained by the agglomeration of a sufficient number of submiliary tubercles. Nodules as large as a pea or even a walnut, are not uncommon. It is only in this broad sense that the miliary tubercle has any specific character, and then only as an "eruptive form."

The definition of tubercles as a new-growth made up of small cells is entirely insufficient. Besides the cellular granulation-like tubercles, such as are met with in inoculated guinea-pigs, there is a fibroid tubercle which I have found most frequently in syphilitic patients, and a lymphadenoid tubercle which occurs as the rule in scrofulous patients. Whether these differences depend upon any real difference in the constitution of the individual, or upon the different ages of the new-growth, or upon some unknown causes, cannot as yet be decided. It appears, too, that the individual constitution not only exerts an influence

on the disposition to become inoculated with tubercle, but also on the character of the tubercle thus produced. I conjecture this not from the lymphadenoid structure, but from the unusual size of the tubercle cells in scrofulous persons. These cells (Schüppel's epithelioid cells) are twice or three times as large as a white blood globule. At the same time they are more highly refractive, their nuclei are sharply defined and often shining. The nuclei may be segmented, but do not go on to form new cells; they simply produce multi-nuclear cells, sometimes giant cells. In this way is produced a peculiar large-celled germinal tissue (Fig. 1), which I designate as a specific product of scrofulous tuberculosis. I do not mean that scrofulous tubercles produce only this large-celled germinal tissue and nothing else. But I consider this tissue to be the acme of the process, which is not always reached. This large-celled tissue usually forms the middle portion of the submiliary nodules, while at their periphery there prevails a small-celled inflammatory growth which gradually becomes continuous with the normal connective tissue.

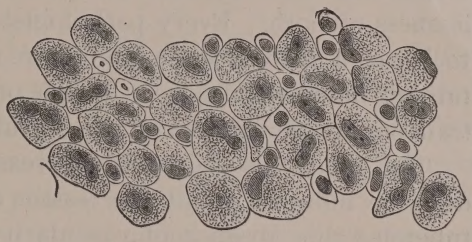

FIG. 1.

Tuberculous infiltration from the floor of a fresh tubercular ulcer of the mucous membrane of the ureter. The small cells are white blood globules, the larger are specifically developed "tubercle cells."

Definition of Tubercle.

Even from what has been already said the tubercle of scrofulous persons might be defined as a small circumscribed focus of scrofulous inflammation, perhaps produced by a minute irritating substance introduced into the tissue. There are, however, other properties of the scrofulous inflammatory infiltrations which are also found in tubercles.

In both there is the same permanent, infiltrating character of the new-growth, the same absence of development, the same tendency to cheesy degeneration. It is well known that the translucent gray miliary tubercles become opaque and cheesy,

first at their centres and then throughout. In the interior of such nodules I have often observed a sort of stratification, as if their substance was twisted around a somewhat eccentric point, or around an axis running diagonally through the nodule. This appearance appears to me rather the result of pressure than the expression of intended organization. I mention it because we often find it very well marked in scrofulous infiltrations which have become cheesy, and in the tubercles of the lungs.

The most important point of similarity, however, between human tubercles and scrofulous infiltration is the absolute bloodlessness of both. Every pathologist knows that it is impossible to inject a real tubercle, and there are never blood-vessels containing blood within one. This is one reason for the pale, semi-transparent appearance of a fresh tubercle.

The same absence of blood-vessels will be noted when we describe more in detail the lesions of phthisis. In the miliary tubercles this absence of vascularity is especially important in relation to their connection with the blood-vessels and the manner in which they are developed.

The Origin of Tubercles.

It has already been taught by many that the vascular connective-tissue system of the body is in general the tissue in which tubercles are developed. Within this system we must distinguish mobile and stable cellular elements. To the mobile elements belong the blood globules and the wandering cells; to the stable the endothelium of the blood-vessels and of the lymphatics, the epithelium of the serous membranes, and the fixed connective-tissue cells. It is from these latter elements, from the fixed cells of the vascular connective-tissue system, that the miliary tubercles, in my opinion, originate. Many of the more recent authors regard the formation of a giant cell as always the first step. But I believe that a giant cell is nothing but an endothelial or a connective-tissue cell enlarged, and with an increased number of nuclei. I have, of course, in my studies of tubercle constantly met with these giant cells, and have often used them as a sort of sign-post. In the smaller

tubercles they are usually at the centre, in the larger at the periphery of the nodules. But no special reliance can be placed on them. They occur in many non-tubercular new-growths, and may then divide into groups of smaller cells, without any change in the character of the new-growth. I prefer therefore to derive my convictions of the initial stage of tubercles from the study of such tissues as the omentum majus. There we can see the smallest tubercle as a nodular swelling of a single, non-vascular, connective-tissue trabecula, and that this swelling is due entirely to a growth of the fixed connective-tissue cells in, and of the endothelial cells on, the trabecula.

This minute discussion is of importance in reference to our general comprehension of tuberculosis. When we find that the specific products of scrofula are developed with the aid of the permanent cells of the organism, it strengthens our belief that scrofula is a disease of the entire vegetation. So when we see that a miliary tubercle is a minimal inflammatory focus excited by the resorption of some irritating substance, we naturally look for some determined cellular substratum for the local irritation, and this can only be found in at least one permanent connective-tissue cell.

The determination of this point is of importance in still another way. The permanent cells of the vascular connective-tissue system are arranged continuously. It is well known that the capillaries of the blood and lymph systems are not homogeneous, but composed of a continuous layer of flattened cells, and that the connective tissue cells are direct processes of the capillary walls. When, however, a formative irritation begins, this continuity is broken. The endothelial cells are separated and multiplied. The lumen of the blood and lymph capillaries is thus more or less completely filled up by their proliferating endothelium.

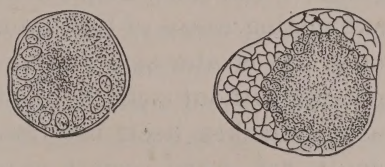

FIG. 2.

Cross sections of two capillary vessels, containing giant cells, from the neighborhood of a tubercle granulum of the lung. × 1 : 300

In Fig. 2 are seen the lumina of two capillaries of the lung, from the neighborhood of a tubercle granulum. Large

giant cells springing from their walls have nearly filled them. We usually find all the endothelial cells of the capillaries adjoining tubercles in the condition of proliferation, and partly obliterating the lumen of these vessels. A good artificial injection of the blood-vessels helps us to follow out more clearly such occluded vessels. It is this obliteration of the blood-vessels which plays a large share in producing the bloodlessness of tubercles already spoken of.

When a tubercle has passed the first stage of its development, it becomes of itself an irritant for the connective tissue in which it is situated. From this time on, therefore, the products of ordinary inflammation become mixed with the specific tubercular products. But even in these mixed processes the cells have the same tendency to reach a large size.

Conclusion.

Tuberculosis in man depends in general on scrofula. Concerning the nature of scrofula we are still very ignorant. One fundamental ingredient of the disease seems to be a misproportion between the volume of the blood and the weight of the body. With this condition exists an abnormality of the entire vegetation, which is especially evident in the course of any inflammatory processes which may arise. Scrofula may be the result of bad food, damp climate, or at least damp dwellings, of exhausting losses of blood and vitality, and is eminently hereditary. It is almost an exception to find a child of scrofulous parents without some taint of the disease. Hereditary scrofula usually makes itself manifest at two periods of life: in early youth, before the seventh year, and again after puberty, between the ages of twenty and thirty years. The other years of life are not by any means exempt from the disease, but it is important to remember that between the ages of ten and fifteen years there is a period of comparative immunity. It is during that period that we may hope by care and treatment to obliterate the traces of previous scrofulous lesions and to prepare against future ones. Upon what this periodicity depends it is hard to say. I look for its cause in a disproportion, during the development of the

individual, between the volume of blood and the weight of the body. Even in the local affections of scrofulous persons there is always a disproportion between the inflamed parenchyma and the absence of the proper congestion of the vessels. Thus the best treatment for scrofula is to increase the volume of the blood, and the best treatment of a scrofulous infiltration is to produce a moderate local hyperæmia.

The great danger in scrofula lies in the possibility of the supervention of tuberculosis.

Like certain animals—guinea-pigs, rabbits, and monkeys—scrofulous persons only need to have some local inflammation set up in order to become tubercular. The tuberculosis depends on the resorption of the individual's own inflammatory products. There is an eruption of numerous metastatic inflammatory foci—the miliary tubercles—while at the same time the patient's strength is rapidly consumed by the cachexia due to the same resorption. Let us assume that in the primary inflammatory focus a special tubercular poison is elaborated, which, besides its general effect on the inoculated person, exerts a direct inflammatory irritation on the inner surface of the lymphatics and blood-vessels. Such an assumption would be supported by our experience of the formation and spread of miliary tubercles. The tubercles are, as a rule, most numerous in the neighborhood of the primary inflammation. Here they are situated in the connective tissue, in the lymphatics, and form an important portion of the phthisical lesions of the different viscera. On account of the histological identity of the scrofulous and tubercular new-growths, it is often impossible, in a given tubercular lesion, to determine how much is inflammatory and how much is tubercular. In the following pages I will show that ordinary pulmonary phthisis exhibits such compound lesions of scrofulous inflammation and tubercular new-growths.

A.—CHRONIC TUBERCULOSIS. PHTHISIS.

II. THE COMMENCEMENT OF THE DISEASE.

Catarrhal Bronchitis and Tuberculosis.

It is an old axiom, but one not now fully recognized, that pulmonary phthisis usually begins with a neglected catarrhal bronchitis. It is certain that many individuals complain at first only of an irritative cough, which has remained after a catarrhal bronchitis. The attending physician finds usually at this time a catarrh of the apex of one or both lungs. At this same time the anatomist would probably find a combination of two conditions: a circumscribed catarrh of the small bronchi at the apex, and an eruption of miliary tubercles in the acini belonging to these bronchi. I have been compelled to this conclusion because in my studies I have never seen a circumscribed catarrh of the small bronchi without an initial tubercle granulum, nor an initial tubercle granulum without some bronchial catarrh. I believe, indeed, that the catarrh is the earlier, the tubercle the later process, and will speak of this point again later. At present it is more important to study the catarrh and the tubercle granulation, and thus at once to appreciate the two most important ingredients of the early stage of pulmonary phthisis.

The Tubercle Granulum.

As we can only speak properly of a tuberculosis of the lungs after an eruption of undoubted tubercles has taken place, we must naturally first consider such an eruption. The first invasion of tubercles exists when we find in the cut surface of a portion of lung, still containing blood and air, numerous white, firm, dry nodules. The form and the size of these nodules vary. Most of them consist of three or more lobules. Here and

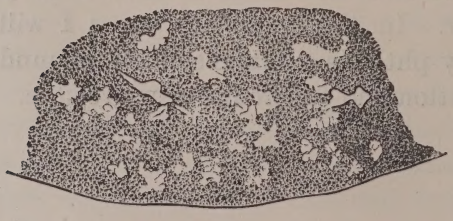

FIG. 3.
Fresh tubercle granulation of the lung.

there they are aggregated into irregular angular figures, or are arranged in groups around the dilated ends of small bronchi filled with pus. If we make thin sections from such a surface we can see how the same nodule varies in shape and size, and how the nodules are related to the ends of the bronchi. In this way it is gradually made evident that "a clover-leaf-shaped nodule, a millimetre and a half in diameter, attached to the smallest visible bronchiole, like a berry to its stalk," represents the typical initial stage of the disease.

In fact, the points at which the smallest bronchioles become continuous with the acini of the lungs are the situation of this first eruption of veritable tubercles.

What are the Acini of the Lung?

All the text-books speak of the lobules of the lung. The lobules are small portions of the parenchyma of the lung; each lobule communicates with a single bronchus. The lobuli are held together, and at the same time separated from each other, by loose connective tissue rich in blood-vessels. This connective tissue is continuous with the connective tissue around the bronchi, the blood-vessels, and the glands, and with that beneath the pleura; it is nourished by branches of the bronchial arteries. In all lungs except those of children, the edges of the lobules can be seen marked out beneath the pleura by black lines of pigment. The general similarity of these lobules seems to be one reason why they have so generally been considered as the ultimate anatomical units of the lungs. There are, however, really acini in the lungs, which combine so that from two to twenty of them form a lobule. If the lung is macerated it is easily seen that the lobules vary much in size. The peripheral ones are tolerably uniform, but those within the interior are very irregular. There is, however, an anatomical element constant both in its form and size, which was first described by Franz Eilhard Schultze (Stricker's Histology) under the name of "Alveolar Passage System." It has the form of a cone-shaped berry, about three mm. long, and of the same breadth (Fig. 3a). The stem of the berry is a single, ultimate, terminal bronchiole, about one-third of a millimetre in diameter. This bronchiole terminates abruptly at the point where it enters the acinus. I have demonstrated that at this point the circular muscular fibres are increased so as to form a sort of sphincter. From here the walls and the lumen of the bronchiole become continuous with the wall and lumina of three diverging alveolar passages, of which the shorter

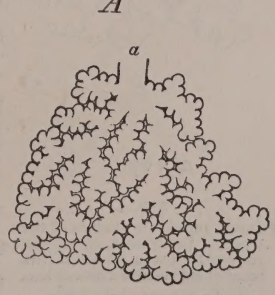

FIG. 3a.
Acinus of the lung, enlarged ten times; at *a* the entrance of the bronchiole.

side branches may well be called infundibula. The alveolar passages are surrounded from place to place with circular and diagonal muscular fibres, which project into their lumina, and thus form the separating angles of each pair of adjoining alveolar septa. In the portion of the walls of the alveolar sacs which are bulged outwards I have not found any muscular fibres.

The relation of the terminal branches of the pulmonary artery to the acini is an additional evidence of the propriety of considering Schultze's alveolar passage system as acini of the lung. These branches pass close by the terminal bronchus into the acinus, and at the point where the three alveolar passages diverge, separate into a large number of branches, which run towards the middle of the acinus, and then break up into capillaries. They run, therefore, within the acini, while the terminal veins are between the acini, so that the blood in the lungs, as in the kidneys, flows from the middle and root of each acinus outwards.

In the third edition of my hand-book of pathological histology I have spoken of the isolated course of the smallest branches of the pulmonary artery with reference to embolism. All these things are of great importance for the right understanding of the manifold lesions of pulmonary phthisis.

The first lesion in pulmonary phthisis is a tuberculous infiltration of all the angles and projections situated at the points where the smallest bronchioles become continuous with the acini. In this way circumscribed white nodules are formed, which we will call, after Laënnec, tubercle granula.

FIG. 4.
Primary tubercle granulum of the lungs. Infiltration of all the connective-tissue edges at the point of entrance of a bronchiole.
a, An enlarged bronchiole.

Let us imagine that we are actually passing from a terminal bronchiole into the respiratory parenchyma. We will be surrounded by the circular edge of the bronchus, in front of us will be a number of sharp, concave edges, which separate the orifices of the three principal alveolar passages, and the infundibula from each other. In these edges are hidden delicate muscular rings and elastic fibres; behind are the terminal branches of the pulmonary artery, surrounded by small lymphatics containing more or less black pigment. The connective tissue which holds together all these tissues, and forms the angles, becomes the seat of tubercular inflammation. It becomes swollen into a hard, white, grayish nodule, with complicated contours, usually lobulated, less frequently jagged.

In this way is formed the primary tubercle of the lungs, Laënnec's tubercle granulation. Fig. 4 is only schematic, to explain what has been stated. Fig. 5, however, is drawn with the camera lucida, and represents accurately the section of a tubercular nodule two mm. in diameter. In it can be seen

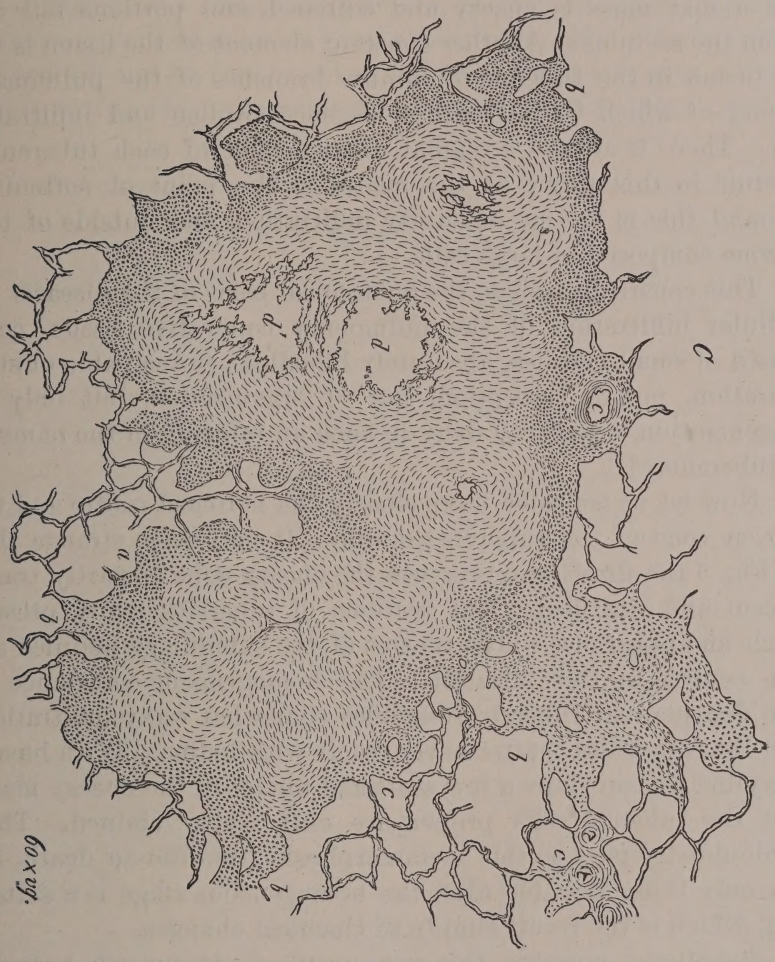

FIG. 5.

A primary tubercle granulum of the lung. Section magnified sixty diameters.

aa, neighboring alveoli. *bb*, spread of the tuberculous infiltration in the connective-tissue septa. *cc*, pulmonary arteries with infiltrated adventitia. *dd*, Cavities formed corresponding to the lumina of alveolar passages.

those anatomical peculiarities, magnified sixty diameters, which are constant in tubercle granulations. Thus we observe in the

separate lobules, of which the nodule is made up, small openings at *d*, *d'*. These openings correspond with the lumina of diseased alveolar passages. Such cavities appear round while the swelling of the wall has not reached its acme ; slit-shaped when it is complete ; as large irregular cavities when the surrounding tubercular mass is cheesy and softened, and portions fall out from the sections. Another constant element of the lesion is the inclusion in the tubercle of minute branches of the pulmonary artery of which the adventitia becomes swollen and infiltrated (*c*). There is always a regular arrangement of each tubercular nodule in this way : at the centre is the point of softening, around this is a zone of cheesy infiltration, and outside of this a zone composed of large cells.

This constitutes, then, the anatomical basis of the disease. A cellular infiltration of the pulmonary connective tissue, composed of small nodules, absolutely bloodless, not capable of suppuration, nor of resorption, nor of organization, but only of degeneration,—who can deny to such an infiltration the name of "tubercular?"

Now let us consider more closely the softened centre and the cheesy zone of the tubercle granule. It may seem strange that in Fig. 5 the drawing represents the cheesy zone as partly translucent and stratified. But sections of tubercles really present such an appearance. The centres of the submiliary nodules are the centres of this striation. The homogeneous substance of the degenerated tubercle resembles somewhat waxy infiltration, but the tubercular matter never reacts with iodine. With hæmatoxyline we can stain a few scattered nuclei in the cheesy mass, but the tubercle cells proper are only feebly stained. They undoubtedly pass in this metamorphosis from life to death, for the only thing possible after the homogeneous stage is a softening, which is the result simply of chemical changes.

Sometimes, however, this metamorphosis is somewhat altered by the deposition of the salts of lime in the cheesy masses. Generally we must assume that when the cheesy tubercle substance remains in a condition of almost absolute dryness at a temperature of from 37° to 39° Celsius (99° to 102.5° Fahr.), there takes place a sort of digestion of the albuminates, such as we

have learned to know from Eichwald's studies on the colloid substance of ovarian cysts. When these albuminous substances have become soluble, they absorb from the surrounding tissue sufficient water for them to become fluid. Thus are formed, first in the oldest, central part of the tubercles (Fig. 5, *d*, *d'*) cavities filled with fluid. This fluid contains the granular detritus which has not been dissolved, and thus looks like pus. If the cavity corresponds to the lumen of an alveolar passage, it may contain pus globules which have wandered in from the bronchi.

Erroneous Explanations of the Tubercle Granule.

I feel only too strongly the contrast between the representation which I have just given of phthisis and the current views on the subject. I believe that Carswell's plates (Pathological Anatomy, Illustrations of the Elementary Forms of Disease, Tubercle, Plate I.) have contributed largely to the views now generally held in Germany. Who can deny that these plates are true to nature? Every one can recognize the bronchioles of the phthisical lung filled with the products of inflammation, and the white bodies looking like bunches of grapes at their extremities. What else could these grape-like bodies be but infundibula filled also with the products of inflammation. When, in addition to this gross appearance, it was found by microscopic examination that in these white bodies were air-cells filled with desquamative epithelium, what could be more natural than the conclusion that pulmonary phthisis is a catarrhal inflammation beginning in the smaller bronchi—a catarrhal broncho-pneumonia. I have for many years supported this doctrine myself, but I am now fully convinced that it is erroneous. The "Carswell-grapes," as they are called by the English, are not infundibula filled with the products of inflammation, but tubercular infiltrations of the connective-tissue angles at the points where the bronchioles become continuous with the acini.

Catarrh of the Bronchi.

The so-called sympathetic bronchitis of phthisis is at first circumscribed, but may later in the disease extend over the

entire respiratory tract. This extension of the disease is usually effected by repeated attacks of acute and subacute tracheo-bronchitis, which leave successive portions of the bronchial tract diseased.

The bronchial catarrh of tuberculosis is not necessarily accompanied by much hyperæmia or swelling of the bronchial mucous membrane. The tracheal mucous membrane is usually pale, while that of the larger bronchi may be mottled with red, especially around tubercular ulcers. Of the smaller bronchi the walls may be thinned and somewhat dilated.

The most characteristic symptom of the catarrh is the nature of its secretion. This is abundant and rather purulent than mucous in character. In the smaller bronchi, at the autopsy, we find a yellowish thin pus mixed with air-bubbles, or, if the bronchi communicate with cavities, the pus is thinner and mixed with cheesy detritus from the walls of the cavities. In the larger bronchi this purulent fluid becomes mixed with the secretions of the numerous mucous glands, but not intimately mixed. If we collect the patient's sputa in a glass partly filled with water, the pus separates and sinks to the bottom, while the mucus floats on the top.

If we turn now to the question of the priority in time of the bronchitis, and of the formation of tubercle granules, no one can be surprised that in accordance with my general views of the relationship between scrofula and tuberculosis, I hold that the bronchitis is the primary and the tubercles the secondary lesion. I believe that in the catarrhal secretions of a scrofulous person is contained the tubercular poison which becomes inoculated in the edges and corners of the narrowest portions of the bronchi. A similar secretion can inoculate the projecting edges of the larynx, the vocal cords, other points of the respiratory mucous membrane, and may even be swallowed and produce tubercles in the lymphatics of the intestines.

The Apices of the Lungs.

Why is it that the tubercular process usually begins in the apices of the lungs? We can offer some explanation of this fact

if we can show that the catarrhal secretions of scrofulous persons are less easily removed from the bronchi of the apices. For if the secretion does thus remain in these bronchi, it will have time to inoculate the surrounding tissues. Two factors have the principal share in influencing the elimination of the bronchial secretion—the respiratory movement of the lungs and the tenacity of the secretion itself. The more freely the respiratory movements are made, and the more a sufficient hyperæmia is present to mix the thick cellular secretion with serum, so much the less likely will the secretion be to remain in such a part of the lung.

In the first place, I would call attention to the fact that in the ordinary upright position of the body, the weight of the dependent arms is exerted on the upper part of the chest, and must restrict the respiratory movements there. This fact corresponds with the almost exclusive occurrence of phthisis in the human race. We know indeed that one part of the thorax can be moved in excess to make up for the insufficient movements of other parts, and that it is by the diaphragm chiefly that this compensating action is performed. But such compensating action is limited in its amount. The lung is not freely movable throughout its entire extent, it is fixed at the point of entrance of the bronchi and blood-vessels. As a result of this the apices of the lungs, situated above these fixed points, can only feel the compensating action of the diaphragm to a small extent. If the apices of the lungs are bound down by old adhesions, they will of course be still less movable. If indeed the body is held erect, so that the arms hang backwards and are supported by the vertebræ, the weight is taken off the chest. But it is in the feeble persons most disposed to phthisis that the contrary habit prevails. They habitually stoop forward, with their shoulders in front, and the movements of the upper part of the thorax necessarily restricted.

In these same persons it is probable that the secretions at the apices of the lungs are inspissated. According to Virchow the thickness of a catarrhal secretion, especially of the bronchi, is in inverse relation to the amount of blood in the part. It has already been shown that poverty of blood is one of the most

important factors of ordinary scrofula. By poverty of blood we mean that there is not a sufficient amount of blood properly to fill the vessels. If the amount of blood in the lungs is insufficient, in the vessels with rigid walls the blood will gravitate to the lower part of the vessels alone; if the walls are elastic, the blood will be found principally in the lower part of the vessels. From this it follows that in such lungs most of the blood will be in the lower lobes, and least in the apices.

III. HEMORRHAGES AND PLEURISY.

The Initial Hæmoptysis.

Before we follow out the further development of chronic tuberculosis in the lungs, we must stop for a moment to consider two complicating conditions which are of very frequent occurrence. These conditions are the initial hæmoptysis and the well-known predisposition of all phthisical persons to pleurisy.

As regards the initial hæmoptysis the assumptions of an inflammatory hyperæmia, or of a greater fragility of the walls of the vessels, have no very credible foundation. I believe, however, that I am possessed of observations of such a character as will explain this condition.

I have already called attention to the fact that the smallest branches of the pulmonary artery are always involved in the tubercle granules. Perivascular tuberculosis, or tuberculous perivasculitis, has been so often described, especially in the brain, that it is unnecessary for me to speak of the cell growth in the adventitia, or of the fusiform and cylindrical nodules. One point, however, has hitherto been overlooked, the fact that this perivascular growth invades and produces a tubercular degeneration of the middle and inner coats of the arteries.

It is only in Rokitansky's description of tubercular basilar meningitis that I have found any mention of this lesion, or of the possibility of its giving rise to hemorrhages. A thorough study of these changes in the small arteries at the base of the brain is necessary before we can speak with certainty of the same changes in the branches of the pulmonary artery; for in the

lungs the course of the vessels is complicated, many pathological changes are crowded together in a small space, and we can only study them by making an infinite number of thin sections.

The way in which the arteries are invaded can perhaps be better appreciated by a glance at Fig. 6, than from a description. We see here a small artery, of which the middle and inner coats are, at a circumscribed point, broken through by the perivascular tubercular infiltration. The drawing represents the upper

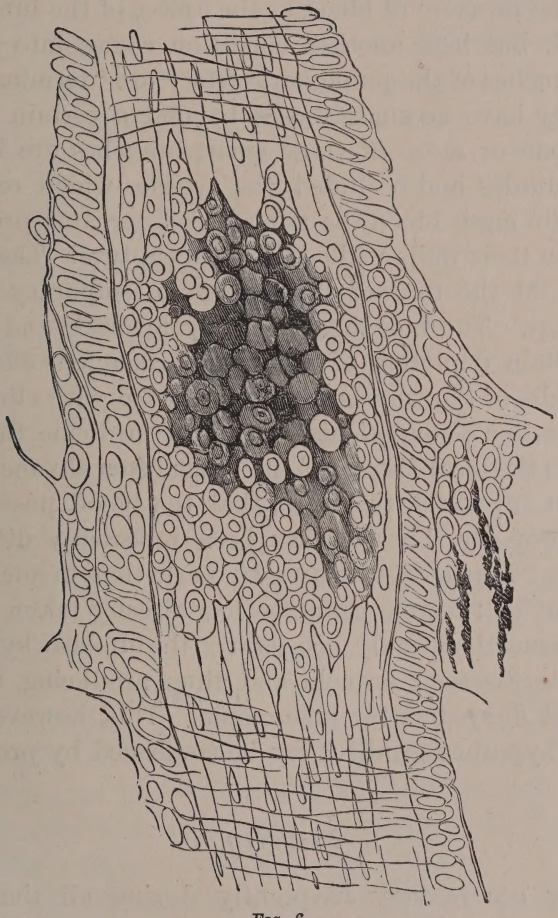

FIG. 6.

wall of the vessel out of focus and unseen, while we look down into the torn and infiltrated depression. At the edges of this depression the inner coat is jagged and irregular, looking as if

changed by maceration rather than by any active process. On the other hand, I have been constantly more and more impressed by the active share taken by the muscular coat. I have even seen an apparent change of the muscular nuclei into epithelioid tubercle cells. But always the wall of the artery is infiltrated with round cells. Even the normal pressure of the blood would seem to be sufficient to rupture the walls of an artery so diseased. In the lungs of tuberculous persons, however, we have already seen that the pressure of blood at the apices of the lungs is below the normal, but here another condition comes into play. The smaller branches of the pulmonary artery are "terminal arteries," that is, they have no anastomoses beyond a certain point. If, therefore, one or more of these small branches are involved in tubercle granules and occluded, the arteries which remain open must contain more blood, be subjected to greater pressure, and any point in their walls will be likely to rupture. There is often, in addition, at the time of the rupture, a temporary congestion of the lungs. The bleeding is purely arterial, and only lasts until a certain depletion of the vascular circle is effected, then the torn walls of the vessels become occluded with clots.

It is a matter of experience in clinical medicine that after an hæmoptysis the local disease of the lungs often advances rapidly. It has been supposed, therefore, that the blood possessed some irritating property, but this has been repeatedly disproved by experiment. I believe, for my own part, that in such cases the development of tubercle granules has already taken place, and that the hemorrhage only accelerates the disease by occluding some of the diseased bronchi and thus producing atelectasis, œdema, and desquamative pneumonia. This, however, remains merely an hypothesis until it can be confirmed by proper autopsies.

Pleurisy.

Pleurisy occurs very frequently during all the stages of tuberculosis of the lungs. It is usually of the adhesive character, producing large masses of new fibrous tissue; later in the disease, with perforation of the lung and pneumothorax, pus is often produced.

Generally speaking, the sympathetic pleurisy is developed in proportion with the disease of the lung parenchyma. That is, over a recently diseased portion of the lung studded with tubercle granules, we find the pleura congested and covered with fresh, soft vegetations. If larger peribronchitic masses have been formed in the parenchyma, there are usually adhesions between the pulmonary and costal pleuræ, and these adhesions become thicker and more extensive as cavities are formed in the peribronchitic masses.

The attempt has always been made to find in these adhesions miliary tubercles, such as are found in tubercular peritonitis, but without success. We do indeed often enough find both costal and pulmonary pleuræ studded with miliary tubercles, but in the ordinary combinations of chronic tubercular lesions in the parenchyma and adhesive pleurisy the pleura is not tuberculous. The pleuritic adhesions do, however, possess a characteristic which throws light on the nature of the pleurisy, namely, the great development of new blood-vessels.

The small irregularities of the surface of the pleura, which can hardly be seen with the naked eye, are little efflorescences of young connective tissue directly connected with the vascular system of the pleura. This, however, requires further explanation. It is erroneous to suppose that the pleural vessels are in any intimate relation with the bronchial arteries. The manifold anastomoses between the bronchial and pulmonary arteries in the walls of the large bronchi have been recently again demonstrated by Hyrtl. But the bronchial arteries and their branches do not disturb the distribution of the pulmonary arteries. In this distribution small arterial branches are regularly given off, which pass directly into the pleura and there branch. They are there distributed alternately with larger or smaller veins, which empty directly into the venæ communicantes (Hyrtl) of the parenchyma. In this way the vascular network of the pleura is intimately and simply connected with that of the lungs. If any disturbance arise, therefore, in the pulmonary circulation, the vessels of the pleura are well fitted to carry off any excess of blood, so long as the venæ communicantes remain free. If, later in the disease, large cheesy masses and cavities have interfered

with the venous circulation, then by the pleuritic adhesions a new outlet may be opened through the intercostal veins. The sympathetic pleurisy has, therefore, rather the character of an inflammatory new-growth than of a simple inflammation.

As already stated, large vascular plexuses form an essential part of the smallest efflorescences of connective tissue. Adhesions between the opposing surfaces of the pleura open short and wide anastomoses between the two sets of blood-vessels. Afterwards, when a larger amount of new connective tissue is formed, the blood-vessels are so numerous that, when artificially injected, they occupy more space than the connective tissue itself.

IV. THE LATER STAGES OF THE DISEASE: CHEESY BRONCHO-PNEUMONIA.

Tubercular Bronchitis and Peribronchitis, Desquamative Pneumonia.

The Crude Tubercle of Authors. Cheesy Broncho-pneumonia.

From the tubercle granules are developed, after a time, the larger cheesy masses, which before Virchow's and Reinhardt's studies were called crude or yellow tubercle. This condition forms a temporary termination of the process. If we follow the development of these masses we will see that even the smallest tubercle granules are increased in size in a definite way. The enlargement is centripetal for each group of granules. To the white berries are joined stems of the same appearance, these stems become united, and form thus larger stellate masses. The commencement of this process of aggregation can be seen in Fig. 3; a later stage in Fig. 7.

Fig. 7.
Cheesy nodules of medium size. Natural size.

If we look at the latter figure more closely, we are struck with the relation of the disease to the bronchi. The form of the cheesy masses cor-

responds to the course of certain bronchi. The masses vary from six to ten millimetres in diameter, and surround the lumina of the bronchi; their external surfaces are irregular. The condition is in fact produced by a spread of the disease from the ends of the smallest bronchi to these smallest bronchi, then to all the branches and the trunk of an intralobular bronchial tree. Nor does the process stop here. The same changes involve still larger and larger bronchi, and only stop when the bronchi with cartilage rings are reached.

As regards the nature of the process, I have no objection to Virchow's designation for it of cheesy broncho pneumonia, but I much prefer to add the name tubercular. There are three factors concerned: 1. A tubercular ulceration of the bronchial mucous membrane. 2. A tubercular peribronchitis. 3. A scrofulous cheesy pneumonia, the so-called desquamative pneumonia. Of these the desquamative pneumonia exhibits the greatest varieties, while the bronchitis and peribronchitis follow a more stereotyped course, like the tuberculosis of the ureters. In both situations we find the same combination of hyperplasia of connective tissue and miliary tubercles. In both situations there is a tubercular ulceration of the mucous membrane, and a secondary tuberculosis of the lymphatics, accompanied by a hyperplastic inflammation of the mucous, submucous, and peri-canalicular connective tissue in which the lymphatics are distributed. We will follow out these changes from within outwards, and commence with the lesions of the mucous membrane.

The Lesions of the Mucous Membrane.

If we follow out with the scissors the ramifications of the bronchi, we will find in the bronchi of about three millimetres in diameter small, white opacities of the mucous membrane, which are rendered more conspicuous by a capillary hyperæmia beneath and around them. As soon as we pass into the still smaller bronchi, measuring two millimetres in diameter and destined for from two to four lobules, the white opacities increase rapidly in number until they occupy almost the entire mucous membrane, leaving only little congested islets between them. At the same

time these opaque spots become thicker until the entire mucous membrane is changed into a thick, yellowish-white layer. The lumina of the intralobular bronchi and of the bronchioles leading to the acini are at first swollen, then as ulceration takes place in the thickening just spoken of, the lumina become enlarged and gradually converted into cavities.

Tubercular Ulceration of the Mucous Membrane.

The histological study of these changes shows that we have here a real tubercular ulceration of the mucous membrane. The whitish opacities are due to a diffuse accumulation of cells between the epithelium and the connective tissue. The little white projections are true tubercles. If we make vertical sections through the smallest of them, we can see that they are composed of single or aggregated submiliary nodules in the parenchyma of the mucous membrane (Fig. 8).

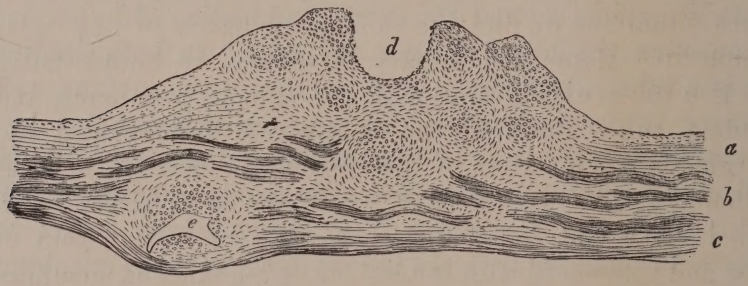

FIG. 8.

Minute Tubercular Ulcer.

a, Mucous, *b*, muscular, *c*, submucous coat. The epithelium is absent. *d*, Loss of substance surrounded by miliary tubercles composed of numerous submiliary nodules. *e*, Cross-section of a lymphatic and of a tubercle of the smallest size. × 60.

The nodule represented here, although not larger than a poppy seed, is made up of at least thirty-six submiliary nodules. In the middle of each of these are a number of the large bodies called by me tubercle cells (Fig. 1), whilst their edges and the tissue between them are infiltrated with lymphatic cells. No capillaries which can be injected or which contain blood are to e found in any part of the infiltration. It is for this reason that the infiltration is so pale, translucent, or opaque, and that the

floors of the ulcers are yellowish white. After these tubercular nodules have existed for some time, the cells become looser, and, without any cheesy degeneration, softening and casting off of the central portions of the nodules take place. In this way a sort of subepithelial tubercular abscess is formed. The tubercle cells appear here in the role of pus globules, but instead of forming an abscess, they make with their masses of protoplasm a solid infiltration—tubercle. Out of this is formed an ulcer; but while in an ordinary ulcer the vascular floor produces a pure, fluid pus, here the floor of the ulcer is covered with a dry, white layer of infiltration. In place of suppuration there follows a slow maceration of this dry, white layer, a sort of putrescence, of which the products are taken up in a thin serum exuded from the surface of the ulcer. There is usually no tendency towards healing, but only to an extension of the infiltration.

As the ulcer broadens, the same infiltration of miliary tubercles takes place in its edges, and produces a thick, yellowish-white, non-vascular layer. As it deepens, new tubercles are formed in its floor, and deeper in the subjacent tissue. We are almost obliged to explain the formation of these deeper, secondary tubercles by an absorption of the tubercular poison from the surfaces of the ulcers on account of the systematic connection of such tubercles with the lymphatics of the connective tissue (Fig. 8, *e*). They are either situated in the walls of the lymphatics, so that their periphery forms part of the edges of their lumina, or they surround the lumen so that it is found in the centre of the nodule. The walls of the lymphatics are infiltrated with small cells throughout their entire extent, so that the impression is produced that the tubercle is only a point more intensely irritated of a lymphatic inflamed through its whole extent, which leads from an ulcer into the submucous and peribronchial connective tissue.

The Peribronchial Lesions.

We turn next to the second factor of the disease—the lesions of the peribronchial tissue. I have already stated that I believe the essential part of this lesion to consist in the propagation of

tuberculosis in the peribronchial lymphatics. In justification of this view I can only say: come and see! Every cross-section of an intralobular bronchus is encircled with miliary tubercles, every longitudinal section shows them strung along it like beads. I trust that no one will say that I confound other things with miliary and submiliary tubercles. I understand perfectly how to distinguish an alveolus, filled with desquamated epithelium, from a solid and histologically homogeneous tubercle, although they may be of the same size and close together. To one accustomed to the study it is easily demonstrated that on an average a third part of the white, dense tissue around the bronchi is composed of aggregated, concatenated, and conglomerated round miliary tubercles. The remaining two-thirds consists of the products of a diffuse infiltration of the connective tissue, which is converted into a dense, bloodless, grayish, semi-translucent mass. Each bronchus is converted into a thick, solid cord, which in cross-section represents the often-described picture of the encapsulated tubercle—a cheesy nodule surrounded by a dense semi-translucent capsule. This capsule, however, is not by any means always converted into connective tissue. The infiltration is the product of a scrofulous inflammation, and, therefore, peculiarly liable to cheesy degeneration.

It is hardly necessary for me to remark that from my standpoint I think there is no great use in separating the peribronchial infiltration, as a single inflammatory product, from true tuberculosis of the surface. Such a distinction, however, is very generally recognized, and may have some foundation. But that it is not a simple hyperplasia of connective tissue is evident from the direct continuity which exists between the peribronchitis and the third factor of cheesy broncho-pneumonia, the scrofulous desquamative pneumonia.

Desquamative Pneumonia.

Desquamative pneumonia, also called catarrhal and cheesy pneumonia, joins together the looser products of tubercular bronchitis and peribronchitis into the compact, cheesy nodules which are the natural products of cheesy broncho-pneumonia.

The degree and the manner, however, in which the desquamative pneumonia takes part in the disease, vary so much in different cases that we may use it as a criterion to distinguish the different species of pulmonary phthisis. In this section we will only consider its histology, which is everywhere the same.

It is certain that in desquamative pneumonia the alveoli of the lung are filled with large epithelial cells, and that these cells are formed by a desquamation and progressive metamorphosis of the epithelium of the lungs. The name of desquamative pneumonia is, therefore, a correct one, although it does not designate the real nature of the process, as we shall see further on.

The pulmonary epithelium is formed, as is well known, from the intestinal glandular germinal layer, and has, therefore, originally the same nature as other glandular epithelium. It is genetically equivalent to the epithelium of the glands of the stomach, of the salivary glands, and of the mucous glands. But the resemblance of the pulmonary epithelium to that of the glands in the fully developed organism is confined to this genetic equivalent. As the alveoli are developed and distended, the epithelial cells become flattened and changed into very thin, homogeneous lamellæ, which, to the number of from three to seven, cover the connective-tissue alveolar wall between the vessels. The superficial capillaries break through the continuity of this epithelium, so that their convexities project uncovered in the air of the alveoli. Thus both the characteristic form and the fundamental signification of cells belonging to the intestinal glandular layer—to act as the agents of nutritive changes between the blood and the outside world—disappear. These nutritive changes—here the interchange of gases—take place directly; the epithelial cells are pushed to one side, and merely form the superficial layer of connective tissue.

So Buhl teaches that these cells acquire both the form and the nature of endothelium, and their pathological changes resemble those of the endothelium of the serous membranes, of the omentum, or of the perithelium of the small vessels of the brain. I have demonstrated years ago the nutritive and formative irritability of these last elements, although here, as else-

where, I have had the misfortune to have published my observations before they could be appreciated, so that I was forgotten when the full time for them arrived. I have lately been able, in a case of tubercular peritonitis, to follow step by step the development of large cells, rich in protoplasm, and containing several nuclei from the endothelium of the omentum, and to see the entire correspondence of these changes with those which take place in the pulmonary epithelium in desquamative pneumonia.

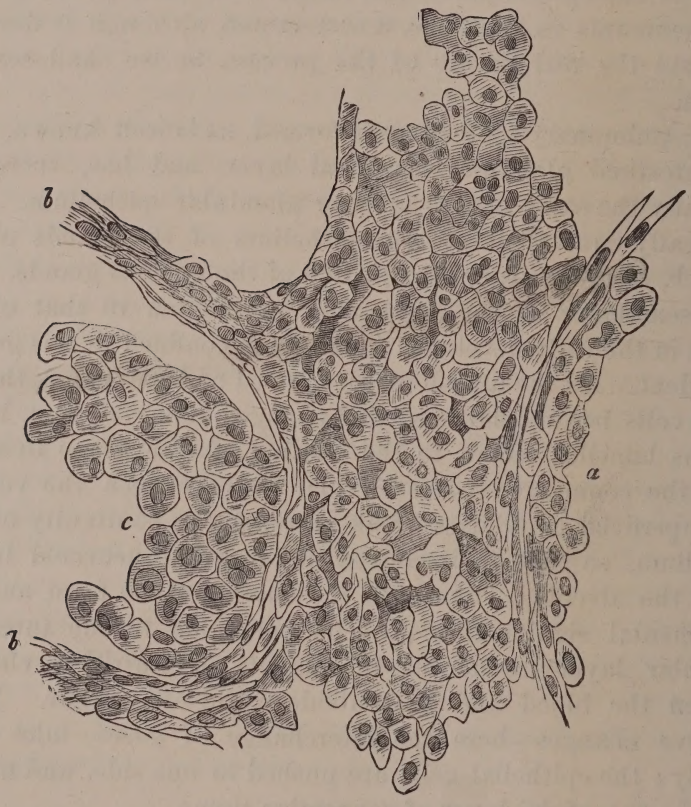

FIG. 9.
Desquamative pneumonia.
a, Alveolar passage. *bb*, Alveolar septa. *c*, Detached pulmonary epithelium.

The cells first become looser, their attached surfaces are covered with a thick layer of finely granular protoplasm, at the same time in each cell the nucleus, which before was hardly visible, becomes swollen and is segmented. Thus are formed large,

granular, epithelial cells, with rounded polygonal contours, and containing one or more nuclei. As it is impossible to demonstrate that a division of the cell bodies follows that of the nuclei, we cannot for the present determine whether these detached cells are capable of multiplying themselves. According to my observations the number of the detached cells in an alveolus is seldom much greater than one would expect to have belonged to the area of the walls from which they had desquamated.

This desquamation and change in form of the alveolar epithelium, however, do not in any case possess the characters of a process belonging to a definite disease, as does the exudation of fibrine in croupous pneumonia. It is rather a stereotyped accident of most of the disturbances affecting the parenchyma of the lung. Hypostasis, œdema, and new-growths of all kinds produce it. This has recently been very clearly shown by Friedländer's experiments on inflammation of the lungs.

In desquamative pneumonia, also, the exudation products of the walls of the alveoli may be considered as merely accompanying the more important changes which are taking place in the connective-tissue parenchyma of the lung. This can be seen in Fig. 9, where are represented the thickened septa between an alveolar passage and two neighboring alveoli partly filled with desquamated epithelium.

The connective tissue is infiltrated with an enormous number of large cells, many of them with two nuclei, nearly all with several surfaces, and flattened. Thus they fill all the spaces in the connective tissue, so that the connective tissue, the elastic fibres, and the blood-vessels can no longer be seen. Only close to the surface of the alveolar passage (at *a*) can be seen a band of smooth muscular fibres, probably a segment of the muscular ring already mentioned. The adjoining alveolar septa (*b*, *b*) are only infiltrated and swollen at their points of junction. Up to this point the vessels are permeable, but no injection will penetrate into an alveolar septum which is completely infiltrated. Whether the endothelium of the blood-vessels becomes swollen, or whether they are only compressed by the infiltration, is undetermined. This absence of blood of course influences the latter stages of the process, and leads to cheesy degeneration.

But before the stage of cheesy degeneration is reached, there occurs regularly another appearance, namely, a peculiar contraction of the infiltrated connective tissue. There is no change of the infiltration cells into fusiform cells, nor any growth of connective tissue, so that the shrinkage is probably only a physical consequence of the infiltration. The contraction may take place while the alveoli are empty, and then they may be obliterated ; but if they have been previously filled with the desquamated epithelium, the alveolar walls and the material within the alveoli, form a dense, homogeneous mass ready for cheesy degeneration. By the naked eye little difference can be seen between the infiltration which is only shrunken and that which is cheesy. Both conditions are usually called cheesy, and with some reason. For while the cheesy metamorphosis of an ordinary abscess is a rare occurrence, dependent on outside causes, and the cheesy spots in malignant tumors are only necroses, the cheesy degeneration of scrofulous inflammation is only the last stage of a regular set of changes, of which the shrinking forms one. It is an histological process of several links, and might be considered as the result of an imperfect, unsuccessful organization.[1]

V. PRINCIPAL VARIETIES OF PULMONARY PHTHISIS.

As I have already mentioned, the manner in which the desquamative pneumonia is developed and combined with the bronchitis and peribronchitis, and the degree of independence which it possesses, determine the principal varieties of pulmonary phthisis. We may distinguish, therefore, a chronic and an acute form of the disease.

The Ordinary Chronic Variety.

The desquamative pneumonia seems to have least of an independent development in the large number of cases in which the scrofulous infiltration of the peribronchial and perivascular

[1] In this point it resembles the scrofulous miliary tubercle, so that when the criterion of its eruptive form cannot be used, we are unable to distinguish the products of miliary scrofulous inflammation from those of the diffuse variety.

connective tissue invades continuously the connective tissue of the adjacent parenchyma of the lung. The desquamative pneumonia then only serves to slowly increase the size of the peribronchitic fibrous and cheesy masses, and to join them together. Thus, in Fig. 10, we can see with the naked eye some small white points which surround the fibrous and cheesy masses. These are the swollen septa of the adjacent alveoli and alveolar passages. Where two of these points are united to form a ring, the wall of an alveolus, or of an alveolar passage, is completely infiltrated, and where, instead of a ring, we find a solid nodule, either their lumina are filled up with desquamated epithelium, or there is present a veritable miliary tubercle. Here and there may be seen the three alveolar passages of an acinus cut transversely, their walls infiltrated and white, and their lumina empty. Sometimes their lumina are not only empty, but dilated, and parts of the alveolar walls have disappeared. This depends on a previous emphysematous condition of the acini. I have found, in fact, that emphysema plays a more important part in the lesions of pulmonary phthisis than is usually believed.

FIG. 10.
Tubercular Peribronchitis. Natural size.

The most marked characteristic, however, of this invasion of desquamation is the way in which it advances laterally into portions of lung previously healthy. Although the peribronchitic infiltration advances steadily from the smallest bronchi to the larger bronchi continuous with them, the desquamative pneumonia advances laterally and invades portions of lung of which the bronchi are still healthy. This explains why in this form of pulmonary tuberculosis the parenchyma next to be invaded can remain permeable to air until parietal infiltration takes place. If such a case remains uncomplicated, there is a steady and regular increase of the anatomical lesions and clinical symptoms.

The Acute Varieties.

The acute forms of pulmonary phthisis are characterized anatomically by a series of pronounced lesions, which are due to the early invasion of the parenchyma belonging to the bronchioles which were primarily diseased. This direction of the disease may be called an increase in a centrifugal direction, in contrast to the centripetal direction of the peribronchitis. But this does not imply that in one set of cases only the one, in another set only the other direction is maintained. On the contrary, we find all sorts of combinations. Usually the question is only whether and when the disease of the parenchyma is superadded to the more insidious process which begins with the formation of the tubercle granule, and goes on with tubercular bronchitis and peribronchitis. This complicating disease is usually prepared for, and induced by, the swelling and occlusion of the smallest bronchi produced in every variety by the tubercle granules and the peribronchitis. The real disease, however, is produced by desquamative pneumonia, which here may properly be called by the name of cheesy lobular inflammation.

Atelectasis.

The question as to what happens if the lumen of a small bronchus is occluded, can be answered by experiments. It is only necessary to cut both pneumogastric nerves in a rabbit's neck, and the paralysis of the glottis thus induced permits portions of the food to pass into the respiratory tract. Portions of grass and cabbage pass into the bronchi of the lower lobes and occlude them.

During the second day the rabbit usually dies. If we then remove the lungs, we find, as a rule, in the lower lobes, along their edges, sharply circumscribed, wedge-shaped foci of which the surfaces are a little depressed below the surrounding surface. These foci are of dark-red color, their pleural surfaces rather bluish. If we make a section, we find these foci unaerated, the large bronchi filled with portions of food, the smaller with purulent secretion. We imagine, therefore, that after the

air is shut off, the corresponding portion of the lung contracts by its own elasticity into the smallest possible compass. The air already present disappears, and is replaced by a certain amount of serum and by the projecting capillaries.

We call this condition atelectasis, like that of a lung which has not yet breathed, yet there is an important difference between the acquired and the non-acquired atelectasis. For since the lung, from the time of the first inspiration, remains distended, and its further development, especially that of the elastic and contractile portions of the alveolar walls, follows another rule than that of the so-called "natural size," when any portions of it return to this "natural size" these same elastic and contractile portions by their shrinkage obstruct the passage of the blood through the capillaries. We see at least that acquired atelectasis changes into other lesions, and may consider that a passive hyperæmia is the cause of the next succeeding changes.

Œdema.

ese changes consist in this, that the atelectatic portions become œdematous. Serum exudes from the vessels, fills the alveoli, and distends them. Here, as in other œdemas, the transuded serum after a time becomes thicker, and the infiltrated portion of lung assumes a gelatinous appearance.

But since the name of "gelatinous infiltration" has been applied by Laënnec to the first stage of diffuse desquamative pneumonia, we will call this later stage of the atelectatic focus simply inveterate œdema.

The dilatation of the vessels diminishes in measure as the alveoli return to their normal dimensions, and the increasing pressure of the intra-alveolar fluid gradually drives out the blood, and the œdematous portion becomes pale, anæmic, semi-translucent, and looks like colloid matter. The nutrition of the lung is, of course, impaired by this condition, and we can often see with the naked eye a peculiar sprinkling of its cut surface, with numerous minute white points, due to fatty degeneration of the alveolar epithelium.

All these conditions, then—atelectasis, recent œdema, invet-

erate œdema, and fatty degeneration—are to be found in circumscribed portions of those lungs in which exists tuberculosis of the small and the smallest bronchi. A special complication is produced if the tuberculous inflammation attacks at an early period the vasa afferentia of the lung acini, in other words, if a tuberculous perivasculitis encroaches on the lumina of the terminal branches of the pulmonary artery. The diminished vascularity in front in such cases produces a congestion of the corresponding veins, and so of that portion of the lung. At first this complication produces a dark-red color of the parenchyma, mottled with hæmorrhagic spots, later a diffuse, black pigmentation.

Slate-Colored Induration.

The parenchyma gradually collapses more and more, the alveolar walls come into apposition with each other, stick together, and form a dense, hard, black, dry mass, traversed by few blood-vessels, and which may well be called *slate-colored induration.* In it the alveolar septa can still be recognized in thin sections in spite of their close apposition. In my opinion there is no reason to think that any inflammation precedes this condition of slate-colored induration.

Cheesy Lobular Masses.

Where, however, a diminished, but still pervious circulation rather favors the development of a desquamative pneumonia, a condition especially induced by the obturation of the bronchi, then the anatomical picture is different. The cellular infiltration of the alveolar walls, and the filling up of their lumina invade uniformly an acinus, or an entire lobule. We

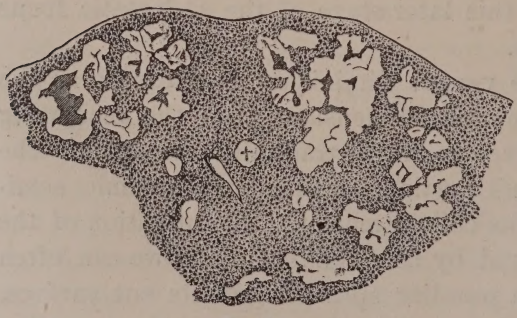

Fig 11.
The explanation of this figure is given in the text.

find then, first, a grayish opacity (infiltration gélatineuse, Laënnec), afterwards this opacity increases, the parts become more and more bloodless, and diffuse cheesy degeneration takes place.

In Fig. 11 we see a portion of lung studded with cheesy nodules of this character. The smaller include each a single acinus, the larger consist of acini united so as to embrace from one to three lobules. The branching of the bronchi is indicated in the cheesy masses by cleft-shaped anastomosing lumina. Instead of these all the larger nodules enclose a cavity due to softening, which here, as in the peribronchitic masses, indicates the beginning of a larger cavity. These nodules are surrounded by atelectatic, œdematous, or gelatinous parenchyma in the preliminary stage of desquamative pneumonia. In this condition the lung gives the same physical signs as if it were in the state of cheesy hepatization, but it differs widely in the fact that the condition is not a permanent one, and the lung may return to a normal state.

Cheesy Lobar Pneumonia.

A still greater degree of independence belongs to that form of desquamative pneumonia which occurs as a diffuse inflammation of an entire lobe affected by tuberculosis. Usually the earlier stages of the disease have already passed by at the apex of the lung, and cavities have formed, when a sudden attack of cheesy pneumonia comes on, and involves at once the rest of the upper lobe. This usually happens with an acute exacerbation of all the clinical symptoms: the fever, dyspnœa, and rapid pulse, so that the impression is given of an intercurrent, croupous, lobular peripneumonia, and the first glance at the lungs might seem to confirm this opinion, for, as Virchow has shown, there are very often in the alveoli plugs of real fibrinous exudation, so that the section of the lung looks granular. But the distinction lies in the infiltration of the alveolar walls, a condition very slightly developed in croupous pneumonia. In scrofulous peripneumonia in all its stages we can recognize, with the naked eye, the thickening and the grayish and whitish infiltration of the alveolar septa.

As the patients frequently die from the subacute, lobular,

desquamative pneumonia, we often have the opportunity to study the different stages of the process. In the first stage the lung is changed into a reddish gray, flesh-like, rather transparent mass, from the cut surface of which exudes a large amount of almost clear, sticky fluid (infiltration gélatineuse, Läennec). Microscopical examination of this fluid shows blood globules; large, rounded epithelioid cells, which formed the large-celled scrofulous infiltration of the alveolar walls; desquamated epithelial cells, sticking together in groups of yellowish color, sometimes containing two nuclei, sometimes brownish pigment. Even at this early stage the blood-vessels are certainly obstructed. At least it is by no means as easy as in croupous pneumonia to obtain a complete injection of all the capillaries. Buhl believes that an arterial perivasculitis accompanies the development of desquamative pneumonia, and induces disturbances of nutrition. "A growth of cells, with small, shining nuclei, accompanying the smallest arterial twigs, situated in their adventitia, sometimes diffuse and sometimes nodular," seems to him to regulate the extent of the second stage of desquamative pneumonia—the cheesy degeneration. I am at present able to confirm Buhl's observations, although not quite prepared to adopt the entire extent of his conclusions.

The cheesy necrosis often begins in separate nodules of the size of a shot. These enlarge, become confluent, and form larger nodules of irregular size. More frequently I have seen a diffuse cheesy degeneration of the entire infiltrated parenchyma. Sometimes the alveolar septa first become paler, and form a stiff framework of yellowish-white, brittle material. The openings of this framework, the lumina of the alveoli and alveolar passages, sink below the level of the section. Sometimes the desquamated epithelium in the alveoli first becomes necrotic. Then we see minute, white granules projecting from a reddish basement substance, until the alveolar septa are involved in the same change, and all is converted into a uniform, whitish-yellow, dry, friable mass.

Our knowledge of the subsequent changes in such a large infiltration, involving a considerable portion of a lobe. is imperfect. It is indeed common enough to find numerous large and

small cavities in a lobe completely infiltrated and cheesy, and we may suppose that such cavities are due to softening and maceration of the necrotic parenchyma. But such a general infiltration is so often developed around cavities already formed, that it is difficult in any given case to tell whether the cavities have been formed before or after the general infiltration.

If we attempt, from what has been said, to form a judgment of the real nature of cheesy desquamative pneumonia, we are compelled to give prominence to two characteristics: first, its close relationship to tuberculosis; second, the permanent character of the inflammatory products infiltrated in the connective tissue. Both lead us to consider desquamative pneumonia as a specific scrofulous inflammation. But we must recognize in it a certain independence of tuberculosis, and be prepared to see it occur as a genuine inflammation. According to Buhl, there is such a genuine desquamative pneumonia, of which the characteristics are: one or more lobes are involved always, including the upper, but seldom both lungs; after the disease has lasted six or eight weeks the volume and weight of the affected lung are increased, its pleural surface is smooth, or coated with a little fibrine; the pleura is swollen and studded with ecchymoses. The parenchyma is brittle, mostly unaerated, the alveolar septa stiffened with infiltration, congested, and pigmented. The brittleness in some places passes into softening. The exuded fluid contains fatty epithelium from the alveoli and bronchi. The bronchial secretion contains fatty epithelium, which may be detected in the sputa, and aid in the diagnosis.

Has no one else seen a case of such genuine desquamative pneumonia? It almost seems so. For myself, I have never seen such diffuse cheesy infiltrations, except in combination with tubercular granules, peribronchitis, or cavities. But I should not be surprised to meet with such an uncomplicated pneumonia as Buhl describes, and would consider it a specific, scrofulous, primary inflammation of the same nature as scrofulous arthritis, orchitis, etc.

VI. CAVITIES.

Definition.

We call cavities all those larger spaces in phthisical lungs which are formed by necrosis and destruction of the parenchyma, and not by dilatation of the bronchi. Bronchiectasia, however, plays such an important part in the formation and development of real cavities, that we must first consider separately that condition.

Bronchiectatic Conditions.

Bronchiectasia plays an important rôle in the earlier stages of our disease, while it is still confined to the apex of one or both lungs. Nothing is more common than to find, at an apex where the process is at a standstill, extensive dilatations of the bronchi. As the disease advances we find them regularly in the freshly granular portions of the lung, larger and more marked in those studded with nodules, and also in those completely infiltrated. The cause of such bronchiectasia lies partly in the greater looseness and extensibility of the inflamed walls of the bronchi, principally in the dilating force which every inspiration exerts on the entire lung. At the earliest stage of tuberculosis the swelling of the passages to the respiratory parenchyma, due to the tubercle granules, causes a dilatation of the bronchi. This swelling is developed gradually, and at many points is never complete, for the lung can be artificially inflated from the bronchi. The catarrh adds to the trouble by still further occluding the bronchi with pus, etc. This is of importance in reference to the physical examination of such patients. We may on one day find the physical signs given by a portion of the lung normal, and on the next day the stoppage of the alveolar passages produces an atelectasis and œdema which give dulness and absence of respiratory murmur, and yet no permanent infiltration is present. But we will speak now of the bronchus in front of the obstructed portion, and not of the parenchyma behind it. The former must necessarily become more and more dilated, for the dilating force, which cannot reach the obstructed parenchyma,

must exert all the more effect on the corresponding bronchi. This dilatation will be the more marked, the less the dilating force can be transmitted to the adjoining parenchyma. Bronchiectatic conditions occur therefore most regularly and most extensively in portions of lung which are situated next to other portions already infiltrated and immovable, and which have themselves recently become the seat of tubercle granulations. Thus we find in Fig. 7 dilatations in two intralobular bronchi, such as are wont to occur in all the bronchi belonging to the same region. We can judge best of these dilatations if we hold the lung in the left hand, and follow out the ramifications of the bronchi with a pair of scissors. We see then that the dilated bronchi have thinner and thinner walls, until there is only a thin wall coated with pus and looking like the pyogenic membrane of an abscess. We can show, however, that this membrane is continuous with the wall of the bronchus, and of the same structure. Carswell's pictures show very well these conditions and the enormous dilatation of which even the smaller bronchi are capable. While we recognize the extent to which bronchiectasiæ are developed and the important share they take in the formation of cavities, we must not fall into the error of considering them to be the only factor, and overlook the share taken by the destruction of the parenchyma of the lung.

The Early Stage of the Formation of Cavities.

I have already shown that the little spots of softening found in the interior of the tubercle granula, and corresponding to the lumina of the alveolar passages, are the commencements of cavities. When the process advances to the bronchi next in size, cheesy nodules are formed as large as a hazel-nut, and comprising from one to three lobules. In these masses the divisions of the bronchi form the plan of the future cavities. Just before the cavities are formed, therefore, we find usually a system of softened spaces, which traverse the cheesy nodule from its root to its periphery in the form of canals filled with granular, yellowish, odorless detritus. The softening has begun in the material filling the lumina of the bronchi, and then has involved the infil-

trated mucous membrane. But the infiltrated peribronchial and perivascular connective tissue for a time resists the softening process. To establish a cavity the only thing now necessary is to empty the softened mass into the bronchi. If we look for the reason why this matter is not at once thus emptied, we will be surprised to find that the lumina of the communicating bronchi, with few exceptions, are wide open. We must then inquire what forces effect the discharge of the softened matter and the enlargement of the cavities, and we will find at once that the mechanism of respiration plays the principal part.

The Opening and Enlargement of Cavities by the Mechanism of Respiration.

The breathing lung is always in a condition of passive distention, even at the moment of the deepest expiration. We must represent to ourselves that the elastic and contractile elements of the lung are arranged in the shape of a tree, of which the trunk is in the root of the lung, the finest branches in the periphery of the organ. This tree is equally strongly stretched in every cross-section of its ramifications, for if this were not the case the equilibrium would be restored by a corresponding change in form of the segment which was least stretched. The same must be the case if any portion of the lung loses its elasticity from pathological changes. Such a portion will be distended beyond its normal size until the equilibrium is restored. So it happens in ordinary catarrhal bronchiectasia. The wall of the bronchus, inflamed, infiltrated, and hyperplastic, is rendered more movable, and therefore less able to withstand the extensile traction of the muscles of inspiration than the surrounding parenchyma. The consequence is, that the bronchus is dilated and the parenchyma contracts.

It would be a still simpler case of pathological alteration of the normal tension of the parenchyma of the lungs, if it were possible to make an incision into a bronchus without producing pneumothorax. We could understand at once how the edges of the wound would contract partly towards the root of the lung, and partly towards the periphery, and in consequence of this

a disproportionately large cavity would be formed. This simple case now is of interest in regard to the formation of cavities.

So long as the infiltrated peribronchial and perivascular connection is still undisturbed, it forms a system of solid radii, which branch from the root through the whole cheesy mass and prevent it from participating in the movements of inspiration. The inspiratory forces exerted on the infiltrated parenchyma are especially transmitted, as we saw, to the corresponding bronchi, which become longer, dilated, and thinner. Finally, however, the destruction of these retinacula is completed. At once the entire bundle is torn off at the root of the wedge-shaped cheesy mass, and hangs in loose fragments in the softened cavities. Now the forces of inspiration can reach the periphery of the softened mass, and the air passes into the cavity of softened matter. The cavities are not, therefore, formed by the emptying of their contents into a communicating bronchus, but the air penetrates first into the softened mass and its discharge is a secondary matter. The same thing takes place in the transformation of softened masses into cavities when the entrance of air is excluded by the swelling and occlusion of the communicating bronchi. Here, also, a time comes when the obstacle breaks down, and in the same way, by the aspiration of air, a large pathological alveolus is formed.

The Enlargement of Cavities.

From this time on the cavity, which often enlarges with astonishing rapidity, is to be regarded as a space full of air and acted on by inspiration. Its further enlargement must then depend on the character of its walls and the degree in which they yield to pressure. If we examine this point more closely, we will see that our comparison of this condition with that of a wound of the lung is a just one. The walls of most young cavities have hardly any power of resistance. Cheesy, half-macerated lung parenchyma can resist passive dilatation as little as can the normal, atelectatic, or gelatinous infiltrated lung tissue which is so often found about cavities. The cavities continue, therefore, to enlarge until they reach the pleura,

which then usually becomes thickened and forms their definite limit. The larger they become, so much the more exclusively will the inspiratory force destined for that portion of the lung be expanded in making them still larger. Like the torn edges of a veil, the remaining parenchyma contracts, and is drawn in partly to the periphery, partly to the root of the lobe. It serves then to fill up all the corners which are left between the convex surfaces of the cavities, and to restore somewhat the contour of the lung. At the root of the lung the large bronchi, with their cartilaginous walls, form stiff stumps, which end as if cut off, and project from one to five millimetres into the lumina of the cavities. In the same place the obliterated branches of the pulmonary artery form a network, with grayish, flattened trabeculæ, which may extend directly across a large cavity—from one wall to the other. Some of the shrunken parenchyma may cling to these obliterated vessels.

There is, however, another method by which the increase in size of cavities is effected—by the junction of adjoining spaces. This may take place by the uniting together of two cavities, or of a cavity and a dilated communicating bronchus. The junction of adjoining cavities is effected according to the known scheme of all unilocularization processes, which have their type in the unilocularization of ovarian cysts. The inclusion of the communicating bronchi is effected by the enlargement of the bronchus until the cavity appears like a gigantic alveolus attached to it. The bronchial mucous membrane is atrophied into a thin layer. If at the same time several cavities open into the same bronchus, there remains finally of the bronchial mucous membrane only an arrangement of bands running over the inner surfaces of the cavities. These processes, also, are under the influence of the mechanism of respiration ; but here the same result is reached by uniform traction from without, as elsewhere by pressure from within, namely, the gradual transformation of such irregular spaces into an approach to the spherical shape. The sphere is that figure which is able with a given surface to enclose the largest area. Every space, therefore, which is acted on by a uniform force, whether from within or without, must tend towards the spherical shape.

In the later stages of the enlargement of cavities, is the existence of any pressure from within to be excluded? A positive pressure would be possible during expiration, if one could suppose that at that time the bronchi leading into the cavity were occluded. Such a supposition is rendered possible by the physical signs existing during life. The surrounding parenchyma, which becomes changed into a thick, hard mass, might then, by its stratification around the convexity of a cavity, exert a centrifugal compression. But I prefer to consider the retraction of the parenchyma as the result of its own elasticity, which exerts itself in the direction of the root of the lung, if the attachment to the wall of the thorax is removed.

The fully-formed Cavities.

The appearance of the inner surface of a completed cavity varies with its age.

Young cavities have often a very irregular conformation. The real space is a sort of cleft between the cheesy nodules and lobules which form the wall, and partly fill the lumen of the cavity. It may also be the case that fragments of this kind are free within a cavity, or but slightly attached to its wall. By the contact of atmospheric air the odorless softening changes into a putrid maceration, forming a stinking, greenish, or grumous matter. If this is mixed with the sputa we find elastic fibres in the little solid particles which sink to the bottom in the spit-cup.

Gradually the inner wall of the cavity cleans off, it becomes smoother, grayish, mottled with points of blood, in places whiter and denser. It then secretes a thin, puriform fluid in large quantities, which, if it accumulates, may become bad-smelling.

Of the other important accidents which depend on the formation of cavities, we will only mention the later hæmoptyses and the perforation of the pulmonary pleura.

Of the hemorrhages, we only refer here to those large hæmoptyses which occur later in the disease. Some of these hemorrhages take place at the time when the cavities are commencing. A corresponding branch of the pulmonary artery is torn away when the inner retinacula of the softened cheesy mass are separ-

ated. Others come from larger vessels, which are left like cords traversing the cavities. At the autopsy we find the source of the bleeding, with the help of water injected into the pulmonary artery, usually in an opening of some size (two millimetres long, and one millimetre wide) in a vessel. It is doubtful whether we have to do with a principal vessel eroded in its continuity, or with a branch eroded up to its point of emergence from the principal vessel. The opening usually gapes widely, less frequently is like a tear.

Perforations of the pulmonary pleura are usually produced by small, quickly formed, subpleural cavities. If a cheesy mass is developed slowly, the pulmonary pleura has time to form adhesions with the costal pleura. But if the cheesy degeneration takes place rapidly, the pleura is involved in the necrotic process, the air escapes into the pleural cavity and leads to an acute, general suppurative pleurisy.

VII. HEALING PROCESSES.

If, now, we turn from the destructive changes which we have thus far been considering, and inquire into the possibilities of recovery and repair, we must understand at once that, from the moment that miliary tubercles have commenced the destructive process, there is never a chance that the lung can return to its normal condition. The best to be hoped for is that the lesions already existing may be rendered innocuous.

It is a widely spread opinion that calcification is a means often employed by nature to render the solid products of pulmonary tuberculosis harmless for the organism. We do, in fact, find peribronchitic and desquamative pneumonic masses, not larger than a bean, converted into chalky masses surrounded by pigmented fibrous tissue, and, besides these, there may be smaller rounded chalk-stones formed in the thickened secretion of dilated bronchi. It must also be admitted that these latter masses may be coughed out. I remember myself one case in which from time to time, after attacks of dyspnœa, fever, etc., a number of small calculi were coughed out. On the other hand, it must be doubted whether calcified infiltrations can ever become movable

except by a renewed process of ulceration. These are usually found at the apices of the lungs, where one or more of them are surrounded by dense slate-colored indurated parenchyma. We have here evidently diseased apices which have become healed very early. An exact knowledge of the manner of this healing I have not yet attained to, but some points concerning it will be indicated in the following pages.

Although we do not underestimate the value of calcification as a method of healing in pulmonary phthisis, yet we would be in evil case if nature did not furnish some other means for a favorable result. *This means consists in a shrinkage of the infiltration, combined with a formation of blood-vessels. These new vessels do not penetrate deeply into the infiltration, but surround it and supply it with constant though scanty nourishment.*

This, so far as I know, is a new view on the subject. Hitherto any healing process of tubercular and scrofulous infiltrations, without cheesy or calcific metamorphosis, has been considered improbable. The idea of another kind of metamorphosis of the new-growth first occurred to me ten years ago, in examining an omentum majus filled with miliary tubercles of a peculiar character. These nodules consisted of a very dense network of short anastomosing fibres, mixed with a number of rudimentary nuclei. They were all of spherical shape, and enclosed in a thin layer of vascular connective tissue.

I did not know at first what to make of this observation. But now that I possess so many observations on healing processes in the phthisical lung, I believe that this condition in the omentum represented a healed miliary tuberculosis. For in the phthisical lung also a fibrous shrinkage of the tubercular and scrofulous infiltration, with simultaneous formation of new peripheric blood-vessels, is the most important method for rendering the lesions harmless. But the process does not consist in enclosing the infiltration in a capsule, as if it were a foreign body; the capsule not only encloses, but nourishes the infiltration. The vascular capsule has the same relation to the non-vascular infiltration as does the perichondrium to cartilage. As an example of this condition I will relate the following case:

A man, aged fifty, died on the seventh day of an attack of typhoid fever. Fourteen years before he had been treated in the same hospital. At that time he had infiltration of the right apex extending down to the third rib, hæmoptysis, etc. From this attack he entirely recovered, followed his business, and during the years just before his death enjoyed excellent health, without any further indications of lung trouble.

At the autopsy there were adhesions over the entire right upper lobe. The pleuritic adhesions over the anterior portion of the apex were five millimetres thick, firm, and very vascular (Fig. 12). They corresponded in extent to that of the portion of lung in which were left the residua of the lung affection existing ten years before. The lung was injected *in situ* by the pulmonary artery with Thiersch's blue, by the bronchi with alcohol. The thing which first attracted my attention, and which is shown

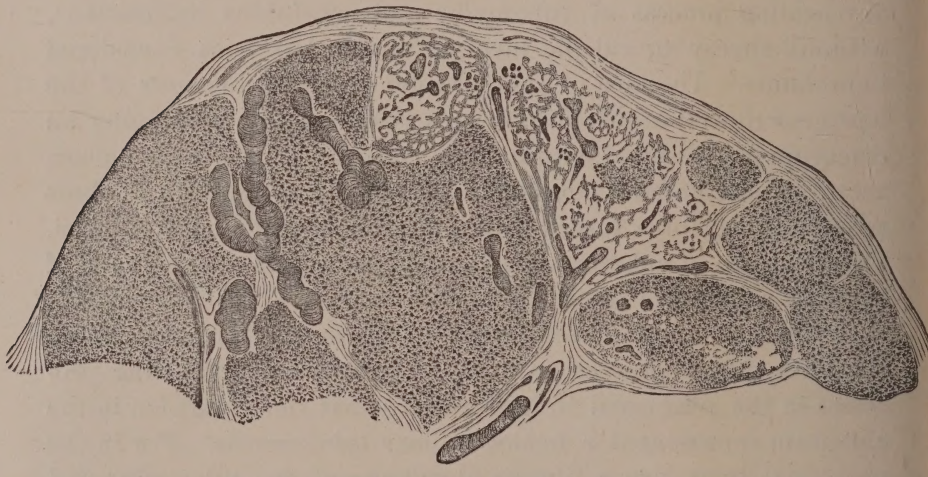

FIG. 12.
Healed Pulmonary Tuberculosis. The explanation is in the text.

in the drawing, was the cicatricial retraction of the diseased lung and the complementary enlargement of the adjacent healthy parenchyma. In this adjoining parenchyma there was moderate dilatation of all the air-passages with atrophy of their walls. The atrophy of the walls, however, only existed in those portions of the lung immediately in contact with the old lesions.

The greater part of the neighboring parenchyma was rather hypertrophied, with an abundant vascular supply.

The old lesions of the lung are surrounded on all sides by a layer of dense connective tissue. This is continuous on the outside with the thickened pleura, on the inside with the peribronchial connective tissue. By the pleuritic thickening and adhesion, and by the thickening of the interstitial tissue the diseased portion of lung is completely surrounded and encapsulated. This interstitial new connective tissue also serves to regulate the circulation, for it is as richly provided with capillaries as are the pleuritic adhesions, and it also contains a considerable number of dilated veins and bronchi (Fig. 12). The lobules and groups of lobules thus encapsulated present a somewhat confused picture. White spots—the previously infiltrated and now shrunken broncho-pulmonary tissue—and blue spots—the emphysematous parenchyma having its vessels filled with the blue injection—alternate with each other. Here is a thickened bronchus, there a dilated one; here again are delicate branching figures looking like thickened lymphatics. Even with the microscope it is only after repeated observations and comparisons that we can obtain much knowledge of the relations of things. If at first we consider some of the lobules which by their form, size, and position can be recognized as such, we find two types of changes which have become stationary. In the one type we find at the centre of a lobule of about the size of a cherry an ectatic principal bronchus, of which the wall is formed of a moderately thick, white membrane. Round about this are hard, white nodules without lumina, corresponding to the accessory bronchi, and imbedded in a parenchyma containing air and blood (Fig. 13, *a*). In the other type we find the entire lobule traversed by a dilated system of bronchi, the parenchyma shrunken together with the walls of the bronchi into a system of thick, cartilage-like septa, and into a similar capsule surrounding the lobule (Fig. 13, *b*). Here and there is a pigmented, indurated, slightly vascular, unaerated parenchyma filling up

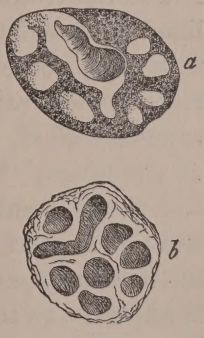

FIG. 13.

the spaces between adjoining nodules. In general, however, the rule holds good that, apart from what remains of the tubercular and scrofulous infiltration, all the other parenchyma returns to a partially normal condition. The air-spaces are filled with respired air and are traversed by a system of vascular connective-tissue septa. How this relatively normal condition can co-exist with the partial occlusion of the communicating bronchi is a second question. It is effected by extensive anastomoses between the adjoining air-spaces, analogous to that which takes place in ordinary emphysema. Finally, there remains only a network with large meshes, its septa formed of parenchyma richly supplied with blood-vessels. As these septa surround small portions of the sclerosed old infiltration, they are able to nourish them, as we have seen is the case with healed miliary tubercles. A few undisturbed bronchi are sufficient, as in emphysema, to keep up the circulation of air around the entire portion of lung formerly diseased. Although the respiratory capacity thus produced is small enough, we must admit that one disease (emphysema) aids materially in effacing the results of another disease (tuberculosis).

It has been truthfully asserted that some cavities may be healed. I have more than once found at the apex, less frequently in other parts, of an otherwise healthy lung, certain small cavities, with dense irregular walls, surrounded by masses left as the results of peribronchitic processes. These cavities are lined with a slate-gray pigmented membrane, and contain a little thin pus, sometimes a few earthy concretions. These are, in fact, healed cavities, and must be distinguished from certain smooth-walled, cyst-like spaces, which also occur at the apices of the lungs. These are lined with a thin slate-colored connective-tissue membrane, covered with ciliated epithelium, and often contain a good deal of muco-pus. They are usually imbedded in thickened parenchyma, and the pulmonary pleura over them is adherent to the wall of the thorax. I believe these spaces to be sacculated bronchiectasiæ, with thin walls.

The fact that we sometimes find an entire upper lobe converted into a single cavity the size of a fist, with thick, dense walls, leads to the belief that possibly larger and complex cavities

may be entirely healed. But in such cases, however chronic, I have never seen an entire cessation of the tubercular process.

Altogether I am of the opinion that the healing of all the local lesions of chronic pulmonary tuberculosis depends on the collateral hyperæmia induced by the obliteration of the normal vessels. By this collateral hyperæmia a new growth of connective tissue and of blood-vessels is effected, which is able to surround with a capsule and render harmless all the products of tuberculosis.

VIII. THE RARER VARIETIES.

All pathological anatomists will agree with me that in autopsies of cases of pulmonary phthisis we find a great variety in the lesions, no two cases being exactly alike. Still, in the majority of cases, we can recognize the different processes already described, and we only wonder at the different pictures produced by tubercle granules and peribronchitis, gelatinous infiltration and cheesy pneumonia, atelectasis, catarrh of the bronchi, bronchiectasiæ, miliary tubercles, tubercular ulceration, pleurisy, interstitial growth of connective tissue, etc. There are, however, a few cases which differ so much from the ordinary ones that it is necessary to mention them somewhat in detail.

In children under five years old the epithelium of the lungs is less atrophied, looser, and more easily detached than in adults. Hence, at this age, in all varieties of pneumonia, we find a predominance of desquamated epithelium in the inflammatory products. This is even true of the idiopathic lobar inflammation of the lung parenchyma, which is analogous to the croupous pneumonia of adults. How much more so of the scrofulous and tubercular affections of the same organ. In fact, desquamative pneumonia plays a principal rôle in the phthisis of children. Even the multiple eruptions of miliary tubercles which attack the liver, spleen, kidneys, serous membranes and lungs, and are identical with the inoculated tuberculosis of guinea-pigs, often grow into disseminated masses of some size, for each tubercle becomes surrounded by a zone of desquamative pneumonia.

According to the size of such zones of pneumonia, the disease

appears as miliary tuberculosis, or as disseminated cheesy hepatization, or as ulcerating lobular pneumonia. I have come to this conclusion especially by comparisons with the inoculated tuberculosis of guinea-pigs. In these animals there are at first only very small miliary tubercles of the lymphatics. To these become associated, with great regularity, small masses of gelatinous, gray infiltration, which become cheesy at their centres.

I assert now, that many cases of cheesy pneumonia in children are due to miliary tuberculosis, and I explain in part in this way the equal predisposition of the entire lung, which is peculiar to the phthisis of children. For the greater number of cases we do not possess such an explanation, excepting cases in which there is cheesy degeneration of the retro-bronchial lymphatic glands, with consequent suppurative periadenitis and discharge of the abscess into the trachea. In such cases we find at the autopsy numerous foci, from the size of a pin's head to that of a walnut, scattered through the whole of both lungs, but most numerous in the lower lobes. These foci present the various conditions of atelectasis, œdema, gelatinous and cheesy infiltration, cavities, and sometimes abscesses. There is probably a direct infection of the lung by the inspired masses of detritus from the open sinuous ulcer. The anatomical changes are partly due to inoculated tuberculosis, partly to the obturation of the bronchi.

Very often in the phthisis of children we find large, wedge-shaped, circumscribed infiltrations of the lung, completely cheesy, separated from the surrounding tissue by suppuration, and existing like foreign bodies in an abscess. Here also belongs the case described on page 638 of the formation of solitary cheesy nodules by scrofulous infiltration of the alveolar septa.

Of the less frequent modifications of pulmonary phthisis which occur in adults, I will mention two characterized by the prominent changes in the lymphatics. We have already seen how admirable a place for development the lymphatics afford to miliary tubercles. The rule is, that the efferent lymphatics from any cheesy focus become the earliest seat of a tubercular eruption. We find the same thing in the case illustrated by Fig. 14. The white masses which we find here infiltrated in the lung are

in unbroken continuity with a moderate-sized cavity in the apex. From this point the infiltration has extended in all directions into the parenchyma, has reached the pleura, and there found a new basis. From this region is taken the drawing. Each one of the white nodules visible here is a so-called encapsulated miliary tubercle. A thick, laminated capsule of connective tissue surrounds the crumbling nucleus, which corresponds to a tubercular infiltration of one of the smallest lymphatics. These nodules are partly scattered in front of the edges of the compact infiltration, or they form delicate chains, or little trees, and conglomerations which appear like branches of the latter. But nowhere, not even where the infiltration is most homogeneous, can anything but tubercle be found in it. The parenchyma is converted into true tubercular new-growth, becomes cheesy, softens, and forms simple cavities. I have found this condition in two cases of constitutional syphilis of many years' standing, and am, therefore, disposed to assume it to be a syphilitic tuberculosis.

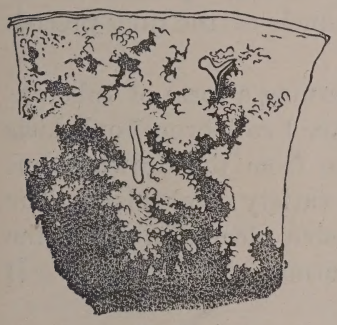

FIG. 14.
Syphilitic (?) lymphangitis nodosa.

The second form of lymphangitis tuberculosa offers a striking resemblance to certain interstitial connective-tissue hyperplasiæ of the liver. But I do not know whether I have the same thing before me which Corrigan describes as cirrhosis of the lungs. A shining, dense, white new-growth marks out the sublobar divisions of the lung. The affection is here seated in those larger tracts of connective tissue which form the root of the lung, accompany and surround the principal bronchi and the larger vessels, and then, when they reach the bronchi of the second order, break up into membranous septa, which enclose the sublobes of the lung. These septa are very rich in veins and in lymphatic vessels. The former run towards the root of the lung, the latter form delicate networks, and are continuous with the lymphatics surrounding the large bronchi.

If now this connective tissue, which corresponds in a measure to the capsule of Glisson in the liver, becomes the seat of a lym-

phangitis tuberculosa, and there is added a chronic desquamative pneumonia, there results the picture represented in the drawing. The larger bronchi and blood-vessels are imbedded in the branching, slate-colored, or black cheesy masses. I have only seen this variety in the lower lobes of the lungs in which there were large cavities in the upper lobes. I consider the entire process to be a tubercular disease of the resorption channels of the lung preceding phthisis proper.

Finally, I will say a few words concerning a form of tuberculosis of the lungs, which, after O. Clarke, I call bronchophthisis pulmonalis. I only know Clarke's case from the drawing, but do not doubt that I have seen the same variety of phthisis. The central point of the disease is an extensive ulceration of all the bronchi of medium size down to the intralobular branches. If

FIG. 15.
Tubercular cirrhosis of the lungs. Upper border of the right lower lobe.

we cut open the lung we notice first numerous canals and spaces, from the size of a pin's head to that of a cherry, which communicate with each other, and traverse the parenchyma in all directions. Each of these spaces is surrounded by a sharply defined white layer, which in some places can be recognized as the ulcerated and hypertrophied wall of the bronchus. In other spaces, especially the smallest ones, microscopic examination shows that what appears as the wall of the little cavity is only

infiltrated parenchyma. These spaces are formed by the ulceration of very small bronchi, with total destruction of their walls. The pneumonia is of the desquamative type. The alveoli are filled with large, granular, epithelial cells, arranged parallel with the surface of the little cavities surrounded by them. The alveoli are flattened in this direction, and distorted into small rhombic clefts.

B.—ACUTE TUBERCULOSIS.

We have shown above that in an invasion of miliary tubercles we can distinguish three stages, as in the invasion of a malignant tumor, namely: 1. The local scrofulo-tubercular primary affection. 2. The tuberculosis of the lymphatic glands. 3. General miliary tuberculosis. General miliary tuberculosis is, therefore, a constitutional disease, and leads to the most severe and fatal disturbances of the general nutrition. The number, size, and distribution of miliary tubercles in any given case do not afford a complete expression of the constitutional effects of the disease. Notwithstanding this, disseminated miliary tuberculosis is not only a valuable sign of the general disease, but it also produces characteristic lesions in the organs in which it is developed. This is especially true of the pia mater and the serous membranes. In these membranes the tubercles act as an irritant which may produce a diffuse inflammatory infiltration.

In acute miliary tuberculosis of the organs of respiration we find both lungs large, congested, somewhat œdematous, emphysematous at the edges of the lobes. Sometimes air escapes into the interstitial tissue by rupture of the small bronchi or alveolar passages. In very anæmic persons (scrofulous children) the congestion is absent, and the miliary tubercles are imbedded in a flabby, collapsed parenchyma, sometimes not even œdematous. The more congested the lungs are, so much the more prominent are the white tubercle nodules on any cut surface. They are usually scattered pretty uniformly through the lung. If we look at them more closely we can see, even with the naked eye, the irregular, jagged outlines of the separate nodules.

With a low magnifying power it is evident that the little projections are infiltrated alveolar septa. We cannot penetrate with any injection to the interior of these stiff, bloodless connective-tissue septa, on the surfaces of which are usually giant cells. In every section of a tubercle we find from five to seven of these mysterious objects, which here are doubtless developed in the lumen of superficial capillaries.

If we examine the interior of these little miliary modules, we find the infiltrated alveolar septa becoming continuous with one, two, or three zones of thickened alveoli. In the middle of these is the lumen of a small branch of the pulmonary artery. The thickened walls of the alveoli here form the essential part of the new-growth. The perivascular connective tissue also participates in the infiltration. Here the new growth reaches, in the formation of groups of large cells, the acme of its development. But there is in this place, except the infiltration of the connective tissue, no special thing to be called a tubercle. The disseminated miliary tubercle of the lung, therefore, is to be defined as a circumscribed scrofulous infiltration. I must be persistent in stating that neither the presence of giant cells, nor the implication of the smallest branches of the pulmonary artery, can alter the definition of the character of such transparent preparations. That miliary tubercles, apart from their etiology, are only small inflammatory foci, and in scrofulous persons only minute foci of scrofulous inflammation, is thus, in my opinion, once more strikingly demonstrated.

INDEX.

Abdomen, appearance of, in pulmonary emphysema, 386; distention of, a cause of hyperæmia of the lungs, 268.

Abortion in croupous pneumonia, 25, 106.

Abscess of the lung in croupous pneumonia, 126; of the mediastinum, its diagnosis from new-growths in the mediastinum, 457; metastatic, in gangrene of the lung, 414; metastatic, their mode of formation, 297, 301.

Acetate of lead in gangrene of the lung, 428; of potassa in pulmonary emphysema, 405.

Acid, benzoic, in the treatment of hyperæmia and œdema of the lungs, 286; carbolic, in gangrene of the lung, 429; inhalation of, 430; muriatic, phosphoric, sulphuric, in the treatment of hyperæmia and œdema of the lungs, 284.

Acids in the treatment of hemorrhages of the lungs, 319.

Acid elixir of Haller, 284.

Acinus of the lung, its definition, 649.

Acute miliary tuberculosis, a complication of catarrhal pneumonia, 197.

Adriani, 271.

Aëtius, 183.

Age in the etiology of acute miliary tuberculosis, 618; of catarrhal pneumonia, 188; of croupous pneumonia, 22; of emphysema of the lungs, 348; of gangrene of the lung, 408; of hemorrhages of the lung, 294; of hypostatic processes in the lungs, 239; of new-growths in the mediastinum, 445; of pulmonary apoplexy, 298; of pulmonary consumption, 505; of pulmonary emphysema, 358; in cerebral and nervous symptoms of croupous pneumonia, 103; in the prognosis of catarrhal pneumonia, 222; of croupous pneumonia, 138; of pulmonary consumption, 596.

Air, cold, a cause of hyperæmia of the lungs, 268; compressed, its employment in pulmonary emphysema, 399; impure, in the etiology of pulmonary consumption, 488; quality of the air to be respired in catarrhal pneumonia, 228; rarefaction of the air in the pulmonary alveoli, a cause of hyperæmia of the lungs, 268.

Albuminuria in catarrhal pneumonia, 211; in croupous pneumonia, 99; in pulmonary emphysema, 374, 382.

Alcoholism, its influence upon cerebral and nervous symptoms of croupous pneumonia, 103.

Alum in the treatment of hemorrhages of the lungs, 318.

Ammonia in the treatment of hyperæmia and œdema of the lungs, 284, 287; anisated spirit of, in the treatment of catarrhal pneumonia, 232; in pulmonary emphysema, 406; muriate of, in catarrhal pneumonia, 230.

Amyloid degenerations in pulmonary consumption, 580.

Anæmia of the Lungs, 264; *etiology*, 264; *pathology*, 264; the bronchi, 264; *symptomatology*, 264; the dyspnœa, 264; *treatment*, 265.

Anæmia, its influence upon cerebral and nervous symptoms in croupous pneumonia, 104.

Anal diseases in pulmonary consumption, 557.

Anasarca in pulmonary emphysema, 374.

Anatomy of tubercles, 642.

Anatomy, morbid, in catarrhal pneumonia, 194; in croupous pneumonia, 40.

Andral, 185, 274, 307, 346, 479, 497, 574, 636.

Aneurism in hemorrhage of the lungs, 293; aneurism, its differential diagnosis from new-growths in the lungs, 442; from new-growths in the mediastinum, 458; from pulmonary emphysema, 395.

Anisated spirit of ammonia in pulmonary emphysema, 406.

Anorexia in catarrhal pneumonia, 203; in croupous pneumonia, 94; in pulmonary consumption, 551.

Antimony, arsenite of, in the treatment of pulmonary emphysema, 396.

Aphthæ of the mouth and throat in pulmonary consumption, 553.

Apneumatosis, see Atelectasis.

Apomorphia, hydrochlorate of, in treatment of hyperæmia and œdema of the lungs, 285.

Apomorphine in the treatment of catarrhal pneumonia, 233.

Apoplexy, pulmonary, its cause, 298; its pathology, 301.

Aretæus, 6.

Arnica in the treatment of hyperæmia and œdema of the lungs, 286.

Arsenite of antimony in the treatment of pulmonary emphysema, 396.

Ascites in pulmonary emphysema, 374, 383.
Asthma in the etiology of emphysema of the lungs, 351; in the symptomatology of pulmonary emphysema, 379; uræmic, in œdema of the lungs, 279.
Atelectasis, 322; *history and bibliography*, 322; definition, 324; *etiology*, 324; congenital atelectasis, 324; collapse of the lung, 324; atelectasis from compression, 326: *pathological anatomy*, 326; congenital atelectasis, 326; acquired collapse, 327; splenization, 328; atelectasis from compression, 329; carnification, 329; patulousness of the fetal openings, 329; *symptomatology and course*, 330; the congenital form, 330; anomalies of circulation, 331; mode of death, 331; the acquired form, 331: the manner of respiration, 331; physical examination, 332; the cough, 332; atelectasis from compression, 332; complications, 333; *prognosis*, 333; the congenital form, 333; the form from compression, 334; *diagnosis*, 334; *treatment*, 335; of the congenital form, 335; of collapse of the lung, 335; emetics, 335; stimulants, 336; other adjuvants, 336.
Atelectasis, a cause of hyperæmia of the lungs, 263; in tuberculosis, 670.
Atrophy and Hypertrophy of the Lungs, 337; *bibliography*, 337; *atrophy*, 337; *definition and etiology*, 337; *pathological anatomy*, 338; general characteristics, 338; the pigment deposit, 339; the bronchi, 339; the heart, 339; *symptomatology*, 340; the dyspnœa, 340; cyanosis, 340; inspection of the thorax, 340; results of percussion, 341; of auscultation, 341; *diagnosis*, 341; *treatment*, 342; *hypertrophy*, 342; general considerations, 343.
Aurelianus, Cœlius, 6.
Auscultation in acute miliary tuberculosis, 619, 623; in atelectasis, 332; in atrophy of the lung, 341; in catarrhal pneumonia, 210; in croupous pneumonia, 36, 82; in gangrene of the lung, 420; in hemorrhages of the lungs, 304; in hyperæmia of the lungs, 277; in hypostatic processes in the lungs, 242; in new-growths in the lungs, 438, 441; in pulmonary consumption, 536, 585, 586, 587; in pulmonary emphysema, 364, 388.
Avenbrugger, 536.
Avicenna, 8.

Baillie, Matthew, 478.
Bamberger, 274, 283, 308, 459.
Banks, 415.
Barrier, 221.
Bartels, 79, 98, 102, 186, 189, 195, 197, 204, 207, 208, 214, 215, 221, 235, 279, 326, 328, 336, 500, 542, 549.
Barth, 83.
Barthez, 138, 186, 413, 500, 504, 549.
Baths during exhaustion of the heart in croupous pneumonia, 176; in the prophylaxis of catarrhs, 227; in the treatment of catarrhal pneumonia, 231; in pulmonary consumption, 607; cold baths as causes of hyperæmia of the lungs, 268; cold baths, their influence upon the temperature in croupous pneumonia, 58; cold baths in treatment of croupous pneumonia, 158; baths of compressed air in pulmonary emphysema, 399; warm baths in treatment of croupous pneumonia, 164.
Baum, 469.
Bäumler, 79.
Bayer, O., 372.
Bayle, 478, 521, 615.
Bed-sores in pulmonary consumption, 563.
Bennet, 445, 468.
Benzoic acid in the treatment of hyperæmia and œdema of the lungs, 286, 287.
Benzoin in pulmonary emphysema, 406.
Berevidge, 440.
Bergmann, 306.
Berkart, 400.
Bertin, 299.
Bierbaum, 436.
Biermer, 214, 349, 351, 354, 376, 379, 387, 389, 395, 399.
Bile pigment in expectoration of croupous pneumonia, 89.
Binz, 168.
Birch-Hirschfeld, 618, 626.
Bladder, caseous affections of, in the etiology of acute miliary tuberculosis, 618.
Blood, changes of, in croupous pneumonia, 1
Bochdalek, 291, 295.
Boerhaave, 8, 307.
Bones, caries of, in the etiology of acute miliary tuberculosis, 617.
Bonjean, 318.
Bonnet, 345, 477.
Boro-tartrate of potassa in pulmonary emphysema, 405.
Borsieri, 9.
Boudin, 507.
Bouillaud, 150, 291,
Bouvier, 168.
Brain, affections of, in pulmonary consumption, 560; local affections of the brain and their influence upon nervous symptoms in croupous pneumonia, 105; œdema of, in croupous pneumonia, 171; hyperæmia of, in pulmonary emphysema, 374, 381.
Brain symptoms in catarrhal pneumonia, 211; in croupous pneumonia, 101; in prognosis of croupous pneumonia, 143.
Brandes, 132.
Brattler, 98.
Brehmer, 611.
Bromine, inhalation of, in gangrene of the lung, 429.

Bronchi, in anæmia of the lungs, 264; in atrophy of the lungs, 339; in croupous pneumonia, 43; in hypostatic processes in the lungs, 240; in pneumonia from embolism, 252, 253; catarrh of, a complication of catarrhal pneumonia, 212; catarrh of, a complication of croupous pneumonia, 110; catarrh of, following gangrene of the lung, 414; catarrh of, in the etiology of pulmonary consumption, 498; catarrh of, in hyperæmia of the lungs, 271; catarrh of, its treatment in connection with pulmonary emphysema, 397; catarrh of, its relation to tuberculosis, 648, 653.

Bronchial casts in the expectoration of croupous pneumonia, 89.

Bronchial glands, affections of, in gangrene of the lung, 415.

Bronchophony in the physical examination of croupous pneumonia, 85; in hyperæmia of the lungs, 277.

Bronchopneumonia, see Catarrhal Pneumonia.

Büchner, 376, 447, 458.

Buhl, 44, 48, 49, 60, 88, 90, 91, 111, 130, 137, 198, 275, 309, 338, 499, 572, 615, 634, 665, 674, 675.

Burkart, 618, 623, 626, 630.

Calcification in tuberculosis, 683.

Camphor in exhaustion of the heart in croupous pneumonia, 175; in the treatment of hyperæmia and œdema of the lungs, 284, 287; in the treatment of pulmonary emphysema, 406.

Cancer in the etiology of pulmonary consumption, 507.

Cancerous growths as causes of hyperæmia of the lungs, 269.

Carbolic acid in gangrene of the lung, 429; inhalation of, 430.

Carbonate of soda in treatment of catarrhal pneumonia, 230; in pulmonary consumption, 605.

Carbonate of potassa in treatment of pulmonary emphysema, 405.

Carbonic acid, discharge of, in croupous pneumonia, 94; increased formation of, in croupous pneumonia, 156.

Carnification of the lungs in atelectasis, 329.

Carriage of the body, in the etiology of pulmonary consumption, 494.

Carswell, 481, 653, 677.

Cartilages of the ribs, changes of, in the etiology of pulmonary consumption, 485.

Casts, bronchial, in the expectoration of croupous pneumonia, 89.

Catarrh, bronchial, in catarrhal pneumonia, 194; in croupous pneumonia, 110; in diagnosis of catarrhal pneumonia, 216; in the etiology of pulmonary consumption, 498; its treatment in connection with pulmonary emphysema, 397.

Catarrh of the stomach in gangrene of the lung, 420.

Catteloup, 122.

Cavities in lungs in catarrhal pneumonia, 197; in chronic tuberculosis, 676.

Cells, epithelial, in expectoration of croupous pneumonia, 89.

Celsus, 6.

Central America, climate of, in pulmonary consumption, 611.

Cerebral form of croupous pneumonia, 126.

Cerebral symptoms in croupous pneumonia, 101; in prognosis of croupous pneumonia, 143.

Chalybeate waters in the prophylaxis of hemorrhages of the lungs, 317.

Chambers, 376.

Charcoal in gangrene of the lung, 429.

Cheesy lobular masses in chronic tuberculosis, 672.

Chest, conformation of, in pulmonary consumption, 485, 507, 531, 596.

Chill, early symptom of croupous pneumonia, 53, 66.

Chilling of the body a cause of croupous pneumonia, 31.

Chills in pneumonia from embolism, 256.

Chloral hydrate, in treatment of croupous pneumonia, 169.

Chloride of sodium in gangrene of the lung, 429

Chlorinated lime in gangrene of the lung, 428.

Chlorine, inhalation of, in gangrene of the lung, 430.

Chomel, 26.

Choroid, affections of, in acute miliary tuberculosis, 621.

Chossat, 565.

Chronic pneumonia, a complication of pleuro-pneumonia, 110.

Church, 451.

Circulation, state of, in catarrhal pneumonia, 201; in croupous pneumonia, 61, 87; in pulmonary consumption, 548; in pulmonary emphysema, 374, 379.

Cirrhosis of the lung in croupous pneumonia, 49; following croupous pneumonia, 130, 182.

Clark, 439, 602.

Clarke, O., 690.

Climate, influence of, in croupous pneumonia, 12; influence of, in pulmonary emphysema, 361; in the etiology of pulmonary consumption, 490, 491; in the prophylaxis of pulmonary consumption, 604; in the treatment of pulmonary consumption, 606; influence of climate of Australia in pulmonary consumption, 491; of Central America, 611; of Costa Rica, 491; of Egypt, 491, 611; of Iceland, 491; of India, 491; of the Island of Marstrand, 491; of Kirghis, 491; of Madeira, 611; of Mexico, 491; of Peru, 491; of Siberia, 491; of South Africa, 491; of South America, 491; of Sweden, 491.

Cloetta, 446, 460.
Cockle, 325.
Cohn, 245.
Cohnheim, 44, 45, 46, 48, 49, 237, 245, 246, 248, 249, 250, 251, 292, 296, 301, 470, 621, 635.
Colberg, 199, 275, 276.
Cold in the treatment of hemorrhages of the lungs, 318.
Cold air, a cause of hyperæmia of the lungs, 268.
Cold baths, as causes of hyperæmia of the lungs, 268.
Cold drinks, as causes of hyperæmia of the lungs, 266.
Collapse of the lung in catarrhal pneumonia, 195, 204.
Collapse in croupous pneumonia, 65; treatment of, 170, 177.
Collenberg, 447.
Conformation of the chest in the etiology of pulmonary consumption, 485.
Complications of acute miliary tuberculosis, 625; of catarrhal pneumonia, 197, 212; of croupous pneumonia, 106, 181; of gangrene of the lung, 422, of pulmonary consumption, 572.
Compression of the thorax in pulmonary emphysema, 404.
Constipation in croupous pneumonia, 96.
Consumption, Pulmonary, 473; *bibliography*, 473; introduction, 475; *history*, 477; *etiology*, 481; the question of inheritability, 482; "sickliness," 483; scrofula, 484; vaccination as a cause of the manifestation of scrofula, 485; influence of the conformation of the chest upon the site of the disease, 485; changes in the cartilages of the ribs, 485; diminished respiratory capacity, 486; bad air and improper food, 488; climate, 490; temperature, 491; elevated position of land, 491; change of climate, 491; deficiency of light, 492; poor quality of food, 492; general influences in early life, 493; the milk, 493; wet-nursing by consumptives, 493; influence of faulty carriage of the body, 494; position of the body during sleep, 495; the experiments of inoculation of tubercles in animals, 495; contagiousness, 497; pulmonary consumption following other diseases, 497; bronchial catarrh as a cause, 498; pneumonia, 499; pleurisy, 500; hæmoptysis, 501; retained decomposing blood, 502; measles, 504; pertussis, 504; diabetes mellitus, 505; the puerperal condition, 505; influence of age, 505; of sex, 506; the connection of malarial affections with phthisis, 506; cancer, 507; goître, 507; emphysema, 507; valvular diseases of the heart, 507; contraction of the chest, 507; *pathology*, 508; *general description and course of the disease*, 508; modifications of the general course, 512; periods of arrest of the disease, 514; the phthisis florida, 514; *analysis of individual symptoms*, 517; the cough, 517; the expectoration, 520; the hæmoptysis, 524; pain, 528; dyspnœa, 529; *inspection of the thorax*, 531; alterations in the form of the chest, 531; anomalies in its movements, 533; results of palpation, 534; of percussion, 534; of auscultation, 536; *laryngeal symptoms*, 543; hoarseness, 543; syphilitic affections, 544; difficulty in deglutition, 546; pain from pharyngeal affections, 546; pain from laryngeal affections, 546; stenosis of the larynx, 547; *symptoms of the organs of circulation*, 548; the heart, 548; the pulse, 548; appearances of the præcordial region during the disease, 548; glandular affections, 549; *the digestive organs*, 551; the stomach, 551; anorexia, 551; nausea, vomiting, and pain, 552; aphthæ of the mouth and throat, 553; thirst, 553; *the intestinal canal*, 554; pain, 554; diarrhœa, 555; anal diseases, 557; diseases of the liver, 558; of the spleen, 559; of the kidneys, 559; of the urinary apparatus, 559; the urine, 559; *nervous symptoms*, 560; the brain, 560; individual peculiarities, 561; affections of the spinal cord and peripheral nerves, 561; *cutaneous symptoms*, 561; changes in the skin, 561; perspiration, 562; eruptions of the skin, 562; bed-sores, 563; œdema, 563; the nails, 563; the hair, 564; the teeth and gums, 564; the emaciation, 565; the fever, 567; its various forms, 567; perspirations, 569; *complications*, 572; the formation of secondary miliary tubercles, 572; pneumonias, 574; pleuritis, 574; meningitis, 575; pericarditis, 576; pneumothorax, 576; acute peritonitis, 578; affections of the digestive organs, 579; amyloid degenerations, 580; of the spleen and liver, 581; of the kidneys, 581; *diagnosis*, 582; the three different forms of pulmonary lesions, 584; *diagnosis of the first form*, 584; percussion, 584; auscultation, 585; the cough and other symptoms, 585; *diagnosis of the second form*, 585; the general rational symptoms, 586; the fever and perspiration, 586; percussion and auscultation, 586; *diagnosis of the third form*, 586; the cough, 587; other symptoms, 587; the differential diagnosis between the different forms, 587; *course and termination*, 591; *prognosis*, 595; influence of parentage, conformation of the chest, age, etc., 596; the cough, 597; the expectoration, 597; hæmoptysis, 598; the laryngeal symptoms, 598; pleuritis, 598; pneumothorax, 598; the pulse, 599; the fever, 599; the loss of weight, 599; symptoms of the digestive organs, 599; affections of the liver, spleen, and kidneys, 600; *treatment*, 600; *the prophylaxis*, 601; marriage, 601; nursing of the infant, 601; his after-rearing in childhood, 602; during puberty, 603; treatment of inflammatory processes, 604; climate, 604; mineral waters, 604; the selection of an oc-

cupation in the prophylaxis, 604; attention to scrofulous symptoms, 604; extirpation of enlarged glands, 605; *treatment of the chronic pneumonia*, 605; general measures, 605; respiratory exercises, 606; residence in an elevated region, 606; the use of mineral waters, 606; the prevention of relapses, 606; inuring the skin to the influences of cold, 607; the use of baths, 607; *treatment of the broncho-pneumonic form*, 607; general measures, 607; spraying of the respiratory organs with saline solutions, 608; the use of fats, 608; inhalation of various remedies, 609; treatment of the fever, 610; treatment of the stage of arrest, 610; influence of climate, 611; revulsive measures, 611; treatment of the laryngeal affections, 611; profuse suppurations, 612; pneumothorax, 612; hæmoptysis, 612; dyspepsia, 612; diarrhœa, 613; perspirations, 613.

Contagion in the etiology of pulmonary consumption, 497.

Cooke, 507.

Copaiba, balsam of, in the treatment of hemorrhages of the lungs, 319.

Copper, sulphate of, in the treatment of hyperæmia and œdema of the lungs, 285.

Cordes, 446.

Corrigan, 689.

Corvisart, 63, 445.

Cough in acute miliary tuberculosis, 622; in croupous pneumonia, 35, 69, 142; in echinococcus of the lungs, 462; in gangrene of the lung, 419; in new-growths in the lungs, 438, 441; in new-growths in the mediastinum, 451; in pneumonia, from embolism, 257; in pulmonary consumption, 517, 585, 586, 587, 597; in pulmonary emphysema, 383.

Cracked-pot sound in croupous pneumonia, 79.

Cream of tartar in the treatment of hyperæmia and œdema of the lungs, 284, 288; in pulmonary emphysema, 405.

Creasote in gangrene of the lung, 429.

Crepitant râles in the physical examination of croupous pneumonia, 82.

Croupous pneumonia, a complication of catarrhal pneumonia, 215.

Cures, grape, milk and whey, in prophylaxis of hyperæmia and œdema of the lungs, 284.

Cyanosis in atrophy of the lungs, 340; in croupous pneumonia, 74; in new-growths in the lungs, 439, 441; in new-growths in the mediastinum, 451, 455.

Daudé, 458.

Dechambre, 339.

Decubitus in gangrene of the lung, 431.

Definition of tubercle, 643.

Delirium tremens, its treatment in connection with croupous pneumonia, 168.

Depaul, 352.

Development, theory of, of pulmonary emphysema, 352.

Diabetes mellitus in the etiology of pulmonary consumption, 505.

Diagnosis of abscess of the lung in croupous pneumonia, 127; of acute miliary tuberculosis, 626; of atelectasis, 334; of atrophy of the lungs, 341; of catarrhal pneumonia, 216; of croupous pneumonia, 134; of echinococcus of the lungs, 464; of gangrene of the lung, 426; of hemorrhages of the lungs, 312; of hyperæmia and œdema of the lungs, 283; of hypostatic processes in the lungs, 241; of new-growths in the lungs, 442; of new-growths in the mediastinum, 457; of pleurisy complicating croupous pneumonia, 109; of pneumonia from embolism, 254; of pulmonary consumption, 582; of pulmonary emphysema, 393.

Diarrhœa in catarrhal pneumonia, 203; in croupous pneumonia, 96; in gangrene of the lung, 420; in pulmonary consumption, 555.

Dibarder, 435.

Diesing, 467.

Diet in treatment of croupous pneumonia, 167.

Dietl, 11, 25.

Digestive organs, symptoms of the, in acute miliary tuberculosis, 625; in croupous pneumonia, 94.

Digitalis in treatment of croupous pneumonia, 164; of hyperæmia and œdema of the lungs, 284, 286, 288; of pneumonia from embolism, 259; of pulmonary consumption, 610; of pulmonary emphysema, 405.

Dinstl, 108.

Diphtheria, catarrhal pneumonia a complication of, 193.

Dittrich, 274, 276, 291, 295, 375, 411, 426.

Donders, 349, 379.

Drasche, 100.

Dress in the prophylaxis of catarrhs, 228.

Drinks, cold, as causes of hyperæmia of the lungs, 266.

Drunkards, croupous pneumonia among, 119.

Duration of catarrhal pneumonia, 220; of croupous pneumonia, 33; of hemorrhages of the lungs, 307; of hypostatic processes in the lungs, 243; of pulmonary emphysema, 391.

Dusch, 469.

Dust, inhalation of, a cause of croupous pneumonia, 31.

Dyspepsia in pulmonary consumption, 612.

Dyspnœa in acute miliary tuberculosis, 619; in anæmia of the lungs, 264; in atrophy of the lungs, 340; in echinococcus of the lungs, 462; in gangrene of the lung, 420; in hemorrhages of the

lungs, 304; in hyperæmia of the lungs, 277; in new-growths in the lungs, 438, 440; in new-growths in the mediastinum, 450, 453; in pulmonary consumption, 529; In pulmonary emphysema, 361, 398.

Eger, 445.
Eichstädt, 469.
Eichwald, 653.
Electricity in pulmonary emphysema, 404.
Elixir, acid, of Haller, 284.
Emaciation in pulmonary consumption, 565, 585, 586, 587.
Emboli as causes of pneumonia, 245.
Embolism, a cause of hyperæmia of the lungs, 268; in the etiology of gangrene of the lung, 410; in pulmonary consumption, 550.
Emerson, J. Haven, 400.
Emetic of Hufeland, 335.
Emetics in the treatment of catarrhal pneumonia, 233; of hemorrhages of the lungs, 319; of hyperæmia and œdema of the lungs, 285.
Emphysema of lung, in catarrhal pneumonia, 196, 208, 216; a complication of croupous pneumonia, 111; in the etiology of pulmonary consumption, 507.
Emphysema, Pulmonary, 344; *bibliography*, 344; *history*, 345; *definition*, 346; *etiology*, 347; heredity, 347; influence of sex, 348; of age, 348; vocation and mode of life, 348, the exciting causes of vicarious emphysema, 348; of substantive emphysema, 349; interlobular and subpleural forms, 351; *mode of development*, 352; vicarious emphysema, 353; effects of inspiratory pressure, 354; the expiration theory, 355; lesions of nutrition, 356; influence of age, 358; lesions of nutrition in the costal cartilages, 359; *general features of the disease*, 360; diseases associated with emphysema, 360; the dyspnœa, 361; the speeech and voice, 361; the influence of climate, 361; the expectoration, 362; the asthmatic form, 362; general course, 362; the heart, 363; percussion and auscultation of the lungs, 364; further progress of the disease, 364; characteristics of vicarious, interstitial, interlobular and subpleural emphysema, 366; *pathological anatomy*, 366; the lungs in the cyanotic cases, 366; in vicarious emphysema, 367; situation of the emphysema, 367; consistency of the lung, 369; its color, 369; the individual alveoli, 369; microscopic appearances, 369, the consequences of emphysema, 373; the heart, 373; the brain and other organs, 374; *relations with other diseases*, 375; with tuberculosis, 375; with heart disease, 376; *analysis of individual symptoms*, 376; the respiration, 376; the difficult expiration, 377; asthma, 379; disorders of the circulation, 379; the heart, 380; cyanosis, 381; hyperæmia of the brain, 381; œdema of the feet, 381; disturbances of the portal circulation, 381; the pulse, 382; the urine, 382; disturbances of nutrition, 382; dropsical effusions, 383; cough and expectoration, 383; *physical examination*, 383; inspection and palpation, 383; various forms of the thorax, 383; appearance of the abdomen, 386; the pectoral fremitus, 386; percussion, 387; auscultation, 388; the cardiac sounds, 389; symptoms of vicarious, interlobular, and subpleural emphysema, 390; *duration and results*, 391; *prognosis*, 392; *diagnosis*, 393; differential diagnosis, 394; from simple dilatation, 394; from pneumothorax, 494; from diseases of the heart and large vessels, 395; *treatment*, 396; emetics and tonics, 396; *prophylaxis*, 396; the bronchial catarrh, 397; the dyspnœa, 398; internal remedies, 398; inhalation of compressed air, 399; the apparatus, 400; modes of employment, 401; compression of the thorax, 404; faradization of the phrenic nerves and abdominal muscles, 404; the asthmatic attacks, 404; cyanosis and dropsy, 405; venesection, 405; diuretics, 405; stimulants, 406; roborants, 406; treatment of interlobular and subpleural emphysema, 406.
Ems, mineral waters of, in pulmonary consumption, 604, 606.
Endocarditis, a complication of croupous pneumonia, 114.
Endocardium, diseases of, complicating croupous pneumonia, 215.
Engel, 291.
Epistaxis in croupous pneumonia, 106.
Ergot in the treatment of hemorrhages of the lungs, 318.
Eruptions of the skin in catarrhal pneumonia, 211; in croupous pneumonia, 100; in pulmonary consumption, 562.
Ether in the treatment of hyperæmia and œdema of the lungs, 287.
Etiology of acute miliary tuberculosis, 616; of anæmia of the lungs, 264; of atelectasis, 324; of atrophy of the lung, 337; of catarrhal pneumonia, 186; of croupous pneumonia, 12, 144; of gangrene of the lung, 408; of hemorrhages of the lungs, 292; of hyperæmia and œdema of the lungs, 265; of hypostatic processes in the lungs, 236; of new growths in the mediastinum, 445; of pneumonia from embolism, 245; of pulmonary consumption, 481; of pulmonary emphysema, 347.
Examination, physical, in acute miliary tuberculosis. 623; in atelectasis, 332; in catarrhal pneumonia, 208; in croupous pneumonia, 74; in gangrene of the lung, 420; in hemorrhages of the lungs, 304; in hyperæmia of the lungs, 277; in hypostatic processes in the lungs, 242; in pneu-

monia from embolism, 257; in pulmonary emphysema, 383.
Expectoration in abscess of the lung in croupous pneumonia, 127; in acute miliary tuberculosis, 622; in croupous pneumonia, 36, 88; in gangrene of the lung, 416; in hemorrhages of the lungs, 305; in new-growths in the lungs, 438, 441; in pneumonia from embolism, 256; in pulmonary consumption, 520, 585, 586, 587, 597; in pulmonary emphysema, 362, 383; inprognosis of croupous pneumonia, 143; promotion of, in the treatment of hyperæmia and œdema of the lungs, 285; in typhoid pneumonia, 124.
Expiration in pulmonary emphysema, 377.
Eye, affections of, in new-growths in the mediastinum, 454, 455.

Fauvel, 195.
Fenwick, 521.
Fever, in acute miliary tuberculosis, 624; in catarrhal pneumonia, 192, 193, 200; treatment of, in croupous pneumonia, 157; in gangrene of the lung, 420; in hemorrhages of the lungs, 306; in hypostatic processes in the lungs, 242; in new-growths in the lungs, 439; in pneumonia from embolism, 256; in pulmonary consumption, 531, 567, 585, 586, 599, 610.
Fever, intermittent, a complication of croupous pneumonia, 119.
Fischer, 418.
Fismer, 55, 58, 59, 66, 107, 116, 119, 124, 126, 134, 139, 161.
Fistula in ano in pulmonary consumption, 557.
Food, improper, in the etiology of pulmonary consumption, 488, 492.
Förster, 370, 434.
Fournet, 538.
Frank, Joseph, 10, 17, 26.
Fränk, J. P., 10, 144, 185.
Fränkel, 503, 635.
Frantzel, 439.
Fremitus, vocal, in the physical examination of catarrhal pneumonia, 209; of croupous pneumonia, 76; of echinococcus of the lungs, 463; of hypostatic processes in the lungs, 242; in new-growths in the lungs, 438, 441; in new-growths in the mediastinum, 452, 456; in pulmonary emphysema, 386.
Freund, 359, 485.
Frey, 340, 343, 375.
Friedländer, 45, 46, 198, 199, 207, 495, 574, 667.
Friedreich, 275, 469, 540.
Froriep, 469.
Fuchs, 355.
Furuncles in catarrhal pneumonia, 211; in croupous pneumonia, 101.

Gairdner, Galen, 477.
Gallic acid in the treatment of hemorrhages of the lungs, 318.
Gamgee, 417.
Gangrene of the Lung, 407; *history and literature*, 407; *etiology*, 408; influence of sex, 408; of age, 408; various causes of its origin, 409; interruption or weakness of the blood-current in the lungs, 409; croupous pneumonia, 409; embolism, 410; emboli from infective purulent centres, 410; gangrene from perforation into the lungs from neighboring parts, 411; from foreign bodies, 411; from stagnant contents of a cavity, 411; from lowering of the general nutrition, 411; from injury, 412; *pathological anatomy*, 412; *the circumscribed form of gangrene*, 412; its situation, 413; the size, form, and number, 413; mode of recovery, 413; bronchitis, 414; transition of circumscribed to the diffused form, 414; hemorrhage, 414; emboli causing metastatic abscesses, 414; affections of the pleura, 414; *the diffuse form*, 415; its general characteristics, 415; *symptomatology*, 415; the sputum, 416; its amount, 416; its characteristics, 416; chemical reactions, 417; microscopic appearances, 417; vibriones and other growths, 418; conditions of putrefaction, 419; the cough, 419; position of the patient, 419; hæmoptysis, 419; symptoms following pleurisy and pneumonia, 420; consequences of swallowing the putrid masses, 420; catarrh of the stomach, vomiting, and diarrhœa, 420; fever, 420; physical examination, 420; *progress of the disease*, 420; the diffuse form, 422; *complications and terminations*, 422; hemorrhage, 422; pleurisy, 422; pneumothorax, 422; rupture through the thoracic wall, 422; causes of death, 423; cases of imperfect recovery, 423; of complete recovery, 424; *prognosis*, 424; influence of age, constitution, and vigor, 424; of causes of the disease, 424; situation of the disease, 425; its size, 425; influence of complications, 425; *diagnosis*, 426; from putrid bronchitis, 426; from abscess of the lung, 427; from pyopneumothorax, 427; *treatment*, 428; *prophylaxis*, 428; improvement of the expectoration, 428; various remedies, 428; inhalations, 429; general treatment, 431; symptomatic treatment, 431; of hæmoptysis, 432; of catarrh of the stomach, 432.
Ganz, 267.
Gases, irritating and irrespirable, as causes of hyperæmia of the lungs, 268.
Geigel, 490.
Geissler, 100.
Geist, 340.
Geographical influences in the etiology of pulmonary consumption, 490.

Gerhardt, 72, 246, 247, 251, 253, 255, 259, 292, 315, 320, 325, 330, 332, 334, 389, 404, 540.
Gerlach, 494, 601.
Gillette, 186.
Glands, bronchial, affections of, in catarrhal pneumonia, 197; bronchial, affections of, in croupous pneumonia, 43; bronchial, affections of, in gangrene of the lung, 415; bronchial and lymphatic, caseous degeneration of, in the etiology of acute miliary tuberculosis, 617; lymphatic, their diseases in relation to scrofula and tuberculosis, 640; affections of the glands, as sequelæ of catarrhal pneumonia, 216; in pulmonary consumption, 549; parotid, inflammation of, a complication of croupous pneumonia, 122; thyroid, affection of, in new-growths in the mediastinum, 455.
Goeden, 118.
Goître in the etiology of pulmonary consumption, 507.
Gordon, 445, 447, 455, 456.
Granulation, absence of, in catarrhal pneumonia, 196.
Graves, 315, 319.
Griesinger, 54, 62, 100, 121, 126.
Grisolle, 18, 20, 23, 31, 32, 34, 51, 52, 64, 66, 67, 71, 76, 89, 90, 104, 108, 116, 117, 121, 122, 123, 125, 128, 130, 135, 138, 139, 140, 186, 574.
Gubler, 89.
Guéneau de Mussy, 352.
Gums, affections of, in pulmonary consumption, 564.

Hæmoptysis, initial, in tuberculosis, 656; in the advanced stages of tuberculosis, 501, 524, 598, 612, 681.
Hæmatoxylon in the diarrhœa of pulmonary consumption, 613.
Hagenbach, 103.
Hair, affections of, in pulmonary consumption, 564.
Haller, 17.
Haller, acid elixir of, 284.
Hannover, 20.
Hasse, 273, 274, 343, 436, 469.
Hauff, 118.
Hauke, 346, 400.
Heale, 271.
Healing processes in tuberculosis, 682.
Heart, in atrophy of the lungs, 339; changes in, in catarrhal pneumonia, 202, 216; affections of, in croupous pneumonia, 43, 63; affections of, as complications of croupous pneumonia, 111; degeneration of muscular tissue of, due to fever in croupous pneumonia, 156; insufficiency of action of, in croupous pneumonia, 153, 157, 169; disease of, influence of, in prognosis of croupous pneumonia, 140; the heart as point of origin of emboli producing pneumonia, 246; diseases of the heart as causes of hyperæmia of the lungs, 269; increased action of the heart a cause of hyperæmia of, lungs, 265; want of power of, a cause of hypostatic processes in the lung, 237; affections of, in new-growths in the mediastinum, 455; diseases of, in the etiology of pulmonary consumption, 507; in symptomatology of pulmonary consumption, 548; in pulmonary emphysema, 363, 373, 380, 389.
Heinze, O. O., 102, 103.
Helber, 449.
Heller, 45, 115, 462.
Hemorrhage, in croupous pneumonia, 90, 106; in gangrene of the lungs, 414, 419, 422.
Hemorrhages of the Lungs, 289; bibliography, 289; *history*, 290; *etiology*, 292; bronchial hemorrhages, 292; vicarious hemorrhages, 293; hemorrhages associated with diseases, 293; influence of sex, 294; of age, 294; hemorrhagic infarctions, 295; their mode of formation, 295; metastatic abscesses, 297; their situation, 297; pulmonary apoplexy, 298; influence of sex, 298; of age, 298; *pathological anatomy*, 298; changes found after bronchial hemorrhage, 298; appearance of the bronchi, 299; the hemorrhagic infarction, 299; microscopical appearances, 300; after-changes in the infarction, 300; metastatic abscesses, 301; pulmonary apoplexy, 301; *symptomatology*, 302; hæmotypsis, 302; the dyspnœa, 304; the expectoration, 305; symptoms of pulmonary solidification, 305; the fever, 306; symptoms of metastatic inflammations and abscesses, 306; of pulmonary apoplexy, 307; *duration, termination, and sequlæ*, 307; duration of bronchial hemorrhage, 307; hemorrhage as a cause or a result of phthisis, 307; course and termination of a hæmoptoic infarction, 310; of metastatic abscesses, 311; of pulmonary apoplexy, 311; *prognosis*, 311; of bronchial hemorrhage, 311; influence of pulmonary disease, 311; hemorrhages in connection with the menstrual flow, 311; hemorrhage in the early stage of tuberculosis, 312; hemorrhagic infarction, 312; *diagnosis*, 312; differential diagnoses, 312; hemorrhagic infarctions, 315; emboli of the bronchial arteries, 316; metastatic abscesses, 316; pulmonary apoplexy, 316; *treatment*, 316; venesection, 316; prophylaxis, 317; preparations of iron, 317; chalybeate waters, 317; treatment during an attack of hæmoptysis, 318; in hemorrhagic infarction, 320; in metastatic abscesses, 320; in pulmonary apoplexy, 321.
Hemorrhagic infarctions, their method of development, 249.
Hermann, 267.
Herpes in catarrhal pneumonia, 211; in croupous pneumonia, 100, 143.

Hertz on anæmia of the lungs, 263; on atelectasis of the lungs, 322; on atrophy and hypertrophy of the lungs, 337; on gangrene of the lung, 407; on hemorrhages of the lungs, 289; on hyperæmia and œdema, 265; on new-growths in the lungs, 433; on new-growths in the mediastinum, 444; on parasites of the lungs, 462; on pulmonary emphysema, 344.
Heschl, 274, 291.
Hewitt, Graily, 331.
Hippocrates, 5, 477.
Hirsch, 13, 16, 17, 477, 483, 490, 506, 507.
Hirt, 31, 490.
History of atelectasis, 322; of catarrhal pneumonia, 183; of croupous pneumonia, 4; of gangrene of the lungs, 407; of hemorrhages of the lungs, 290; of hypostatic processes in the lungs, 236; of pneumonia from embolism, 245; of pulmonary consumption, 477; of pulmonary emphysema, 345; of tuberculosis, 633.
Hoarseness of the voice in pulmonary consumption, 543.
Hoffmann, Fred., 307.
Hopf, 254, 315.
Hoppe-Seyler, 100.
Hörlin, 483.
Horstmann, 447, 453, 456.
Hourmann, 339.
Hueter, 642.
Hufeland, 11.
Hufeland's emetic, 335.
Huppert, 90, 92.
von Huss, Magnus, 14, 17, 51, 72, 95, 104, 107, 110, 112, 116, 119, 125, 128, 130, 133, 182.
Hutchinson, 486.
Huxham, 144.
Hydropericardium in pulmonary emphysema, 383.
Hydrothorax in pulmonary emphysema, 365, 374, 383.
Hyperæmia and Œdema of the Lungs, 265; *etiology*, 265; increased heart action, 265; influence of cold drinks, 266; of cold baths, 268; influence of various diseases, 268; of cold air, 268; of the inhalation of irritating and irrespirable gases, 268; of rarefaction of the air in the pulmonary alveoli, 268; of acute inflammations of the parenchyma of the lungs, 268; of new-growths, 269; of acute and chronic tuberculosis, 269; etiology of passive or obstructive hyperæmia, 269; insufficiency of the mitral valves, 269; disease of the aortic valves, 269; compression of the pulmonary veins by tumors, 269; increased resistance to the heart's action, 269; weakened heart action, 269; *pathological anatomy*, 270; the acute congestion, 270; the chronic form, 270; the general characteristics, 270; splenization of the lung, 271; hypostatic congestion, 271; bronchitis, 271; pulmonary œdema, 271; a result of dropsy, 272; interstitial œdema, 273; the elasticity of the lung, 273; its color, 273; the amount of fluid and its characteristics, 273; condition of the bronchi, 274; the so-called brown induration, 274; hypertrophy of the lungs, 274; *symptomatology*, 276; initial symptoms, 276; the dyspnœa, 277; results of physical examination, 277; later general symptoms, 277; symptoms following œdema, 278; physical examination in hypostasis and hypostatic œdema, 278; œdema a result of a watery condition of the blood, 279; the so-called uræmic asthma, 279; *course and termination*, 281; *prognosis*, 282; *diagnosis*, 283; *treatment*, 284; general measures, 284; prophylactics, 284; passive hyperæmia, 284; acute hyperæmia, 284; venesection, 285; promotion of expectoration, 285; derivation to the skin, intestines, and kidneys, 286; digitalis, 286; treatment of irregularities in the respiratory acts, 287; of chronic passive hyperæmia with œdema, 287; of œdema following cardiac disease, 288.
Hypertrophy of the lungs, see Atrophy and Hypertrophy.
Hypostasis of the lungs, 271.
Hypostatic Processes in the Lungs, 236; *bibliography*, 236; *history*, 236; *pathogenesis and etiology*, 236; diminution in the propelling power of the heart, 237; prolonged maintenance of the body in one position, 237; extent of the process, 237; favoring causes, 237; position of the disease, 237; the question of existence of inflammation, 238; influence of various diseases, 239; of age, 239; development of catarrhal inflammation from these conditions, 239; *pathological anatomy*, 240; splenization, 240; the bronchi, 240; serous effusions into the pleural cavity, 240; catarrhal pneumonic infiltrations, 240; *symptomatology and diagnosis*, 241; general symptoms, 241; fever, 242; local symptoms, 242; termination in resolution, 242; symptoms if catarrhal pneumonia be present, 242; *duration and prognosis*, 243; *treatment*, 243; preventing force of gravitation from acting in one direction, 243; stimulation of the heart's action, 243; diet, 244; exercise of the respiratory muscles, 244.
Hyrtl, 659.

Icterus in croupous pneumonia, 96, 116.
Immermann, 115.
Induration, slate-colored, of the lungs in chronic tuberculosis, 672.
Infarctions, hemorrhagic, their methods of development, 249; their appearance, 299.
Inflammations, acute, of the parenchyma of the lungs, as causes of hyperæmia, 268.

Inheritability in the etiology of pulmonary consumption, 482.
Inselbad at Paderborn, mineral waters of, in pulmonary consumption, 606.
Inspection of the chest, in the diagnosis of catarrhal pneumonia, 208, 217; in croupous pneumonia, 74; in pulmonary emphysema, 383.
Intermittent fever a complication of croupous pneumonia, 119.
Intestines, disturbances of, in acute miliary tuberculosis, 619; diseases of, as sequelæ of catarrhal pneumonia, 216; in croupous pneumonia, 96; derivation to, in œdema of the lungs, 286; affections of, in pulmonary emphysema, 374.
Iodide of potassium in acute miliary tuberculosis, 630.
Ipecac in the treatment of hemorrhages of the lungs, 319; of hyperæmia and œdema of the lungs, 285.
Iron, in the prophylaxis of hemorrhages of the lungs, 317; in the treatment of croupous pneumonia, 177; of hemorrhages of the lungs, 318; in pulmonary emphysema, 406.
Isaakssohn, 371.
Isambert, 275, 276.
Ischl as a sanitary resort, 336.

Jaccoud, 456.
Jackson, 347.
Jacobi, 497.
Jaffé, 130, 411, 417, 418, 425, 429, 470.
Jaksch, 408.
Jenner, 346, 355.
Jörg, 327.
Jortsits, 468.
Juergensen on catarrhal pneumonia, 183; on croupous pneumonia, 3; on hypostatic processes in the lungs, 236; on pneumonia from embolism, 245.
Juniper in pulmonary emphysema, 405.

Kaulich, 408, 448.
Kaupp, 99.
Kidney disease, pneumonia a complication of, 119.
Kidneys, affections of, in acute miliary tuberculosis, 621; in catarrhal pneumonia, 211; in croupous pneumonia, 97; derivation to, in œdema of the lungs, 286; diseases of, in pulmonary consumption, 559, 581, 600; in pulmonary emphysema, 374.
Kiwisch, 541.
Klebs, 494, 601.
Klob, 371.
Kluge, 350.
Koch, 396, 612.
Kocher, 151.
Köhler, 150, 234, 436.
Köster, 574.
Krabler, 220.
Krameria in the diarrhœa of pulmonary consumption, 613.
Küchenmeister, 469.
Küss, 89.

Laennec, 5, 11, 72, 83, 84, 128, 129, 185, 245, 252, 271, 272, 277, 290, 293, 295, 300, 305, 307, 320, 340, 345, 346, 352, 408, 415, 479, 497, 500, 536, 595, 600, 615, 633, 636, 650, 671, 673.
Lambl, 424.
Lange, 399, 439.
Laryngeal symptoms in pulmonary consumption, 543, 586, 587, 598, 611.
Laryngitis in catarrhal pneumonia, treatment of, 234.
Laryngoscopy in the diagnosis of laryngeal or tracheal hemorrhage, 313.
Larynx, stenosis of, a complication of catarrhal pneumonia, 213.
Laurence, 409.
Lead, acetate of, in gangrene of the lung, 428; in œdema of the lung, 287.
Lebert, 31, 50, 54, 57, 59, 66, 67, 96, 100, 106, 116, 124, 125, 266, 276, 279, 286, 287, 294, 309, 312, 375, 376, 389, 408, 409, 413, 417, 434, 437, 439, 447, 456, 466, 480, 571.
Legendre, 324.
Leptothrix pulmonalis in gangrene of the lung, 418.
Leroy d'Etiolles, 352.
Leuckart, 468.
Leudet, 616, 621, 629.
Leyden, 117, 118, 129, 156, 410, 411, 412, 417, 418, 425, 428, 429, 470.
Lick, 630.
Liebig, 102.
Liebermeister, 56, 65, 102, 103, 124, 125, 134, 156, 160.
Light, deficiency of, in the etiology of pulmonary consumption, 492.
Lime, chlorinated, in gangrene of the lung, 428.
Lipmann, 309.
Lippspringe, mineral waters of, in pulmonary consumption, 604, 606.
Litzmann, 469.
Liver, in acute miliary tuberculosis, 621; in catarrhal pneumonia, 203; in croupous pneumonia, 96; in pulmonary consumption, 558, 581, 600; in pulmonary emphysema, 374.
Lobular pneumonia, see Catarrhal Pneumonia.
Lombard, 345.
Look, Theodor, 63.
Louis, 66, 345, 347, 353, 357, 479, 503, 521, 523, 526, 546, 555, 574, 576, 579, 588, 615.
Ludwig, 99, 295.
Lung, acinus of, its definition, 649.
Lungs, diseases of, in the prognosis of bronchial hemorrhage, 311.
Lungs, carnification of, in atelectasis, 329; spleni-

zation of, in atelectasis, 328; cavities in, in catarrhal pneumonia, 197; changes in, after true inflammation in catarrhal pneumonia, 196; changes in, before true inflammation in catarrhal pneumonia, 195; collapse of, in catarrhal pneumonia, 195, 204; consolidation of, in diagnosis of catarrhal pneumonia, 217; emphysema of, in catarrhal pneumonia, 196, 208; affections of, as sequelæ of catarrhal pneumonia, 216; abscess of, in croupous pneumonia, 126; cirrhosis of, in croupous pneumonia, 49; result of croupous pneumonia, 130; gangrene of, a result of croupous pneumonia, 128; gangrene of, a sequel of croupous pneumonia, 182; œdema of, in croupous pneumonia, 171; gangrene of, a sequel of infarction, 253; their condition in hemorrhagic infarction, 252; acute inflammations of the parenchyma of, as causes of hyperæmia, 268; compression of, a cause of hyperæmia, 268; splenization of, in hyperæmia, 271; changes in, in pulmonary emphysema, 366; œdema of, in pulmonary emphysema, 374.

Luschka, 434.

MacCormack, 601.

Malaria, a complication of croupous pneumonia, 121.

Malarial affections in the etiology of pulmonary consumption, 506.

Mangetus, 477, 615.

Manz, 621.

Marck, 399.

Marriage among the predisposed to pulmonary consumption, 601.

Martin, 396, 467.

Massina, 319.

Measles in the etiology of pulmonary consumption, 504.

Meckel, 560, 561, 581.

Membranes, mucous, in tuberculosis, 661; serous, in acute miliary tuberculosis, 621.

Mendelssohn, 346, 355.

Meningitis, a complication of croupous pneumonia, 114; a complication of pulmonary consumption, 575.

Mensuration in the physical examination of croupous pneumonia, 75.

Merkel, 32.

Metallic tinkle in pulmonary consumption, 542.

Miliary tubercles as secondary formations in pulmonary consumption, 572; miliary tuberculosis, acute, a complication of catarrhal pneumonia, 197.

Mohr, 446, 460.

Morgagni, 30, 185, 342, 345, 497, 522.

Morgan, 433.

Morphine, hypodermic use of, in pain and sleeplessness of croupous pneumonia, 168.

Mortality, rate of, in catarrhal pneumonia, 221; in croupous pneumonia, 131, 178.

Morton, 307, 478.

Mozing, 412.

Mosler, 116, 117, 118.

Moutin, 467.

Mouth, affections of, in pulmonary consumption, 553.

Mucine in expectoration of croupous pneumonia, 88.

Muret, 446.

Muriate of ammonia in treatment of catarrhal pneumonia, 230.

Muriatic acid in the treatment of hyperæmia and œdema of the lungs, 284.

Muscles, changes in, in croupous pneumonia, 52.

Musk, in exhaustion of the heart in croupous pneumonia, 176; in the treatment of hyperæmia and œdema of the lungs, 284, 287; in pulmonary emphysema, 406.

Myrrh in gangrene of the lung, 429.

Nails, affections of, in pulmonary consumption, 563.

Narcotics in the treatment of hemorrhages of the lungs, 318.

Nausea in pulmonary consumption, 552.

Nerves, pneumogastric, section of, causing pneumonia, 198.

Nervous system in acute miliary tuberculosis, 624.

Nervous symptoms in croupous pneumonia, 101; in pulmonary consumption, 560.

Neubauer, 97.

Neuenahr, mineral waters of, in pulmonary consumption, 604, 606.

Neukomm, 417.

Neumann, 11.

New-Growths in the Lungs, 433; *pathological anatomy,* 433; fibromata, 433; lipomata, 434; enchondroma, 434; osteoma, 434; cysts, 435; myxoma, 435; sarcoma, 435; osteo-sarcoma, 435; carcinoma, 435; epithelioma, 436; *symptomatology,* 437; the dyspnœa, 438; the cough, 438; the size of the thorax, 438; percussion, 438; the vocal fremitus, 438; auscultation, 438; the respiration, 439; cyanosis, 439; œdema, 439; the pulse, 439; fever, 439; course of the disease, with symptoms of phthisis, 439; *analysis of the several symptoms,* 440; *prognosis,* 442; *diagnosis,* 442; differential diagnosis, 442; *treatment,* 443.

New-Growths in the Mediastinum, 444; *bibliography,* 444; *etiology,* 445; *pathological anatomy,* 446; lymphadenomata, 446; lipomata, fibromata, and osteomata, 446; cysts, 446; carcinoma, 447; sarcoma, 447; situation of the tumors, 447; their size, 448; points of origin of mediastinal tumors, 448; results of pressure, 448; metastases, 450; *symptomatology,* 450; the respiration, 450; pains, 450; the cough, 451; the

voice, 451; head symptoms, 451; nausea, 451; circulatory symptoms, 451; position of the patient, 451; his general appearance, 451; percussion, 452; the vocal fremitus, 452; the heart sounds, 452; *analysis of the several symptoms* 453; pains in the chest, 453; the dyspnœa, 453; head symptoms, 454; dysphagia, 454; affections of the eye, 454; the pulse, 455; the heart, 455; cyanosis and œdema, 455; the thyroid gland, 455; the pleural and pericardial cavities, 455; the vocal fremitus, 456; *duration, result, and prognosis*, 456; *diagnosis*, 457; from abscess of the mediastinum, 457; from aneurisms, 458; from pericardial effusions, 459; from infiltration in the lungs, 459; from pleural exudations, 460; diagnosis of tumors of the posterior mediastinum, 460; *treatment*, 460.

von Niemeyer, F., 53, 74, 171, 172, 173, 266, 268, 276, 292, 295, 308, 341, 351, 353, 359, 377, 386, 388, 393, 405, 502, 634.

Niemeyer, Paul, 166.

Night-sweats in pulmonary consumption, 586.

Noak, 467.

Nursing in the etiology of pulmonary consumption, 493.

Nutrition in the treatment of catarrhal pneumonia, 234; condition of, in croupous pneumonia, 92; disturbances of, in pulmonary emphysema, 382.

Nux vomica in the treatment of pulmonary emphysema, 396.

Obersalzbrunn, mineral waters of, in pulmonary consumption, 606.

Occupation in the prophylaxis of pulmonary consumption, 604.

Œdema of the lungs, see Hyperæmia and Œdema.

Œdema of the brain in croupous pneumonia, 171; of the lungs in chronic tuberculosis, 671; of the lung in croupous pneumonia, 171; in hypostatic processes in the lungs, 242; in new-growths in the lungs, 439, 441; in new-growths in the mediastinum, 451, 455; in pulmonary consumption, 563; in pulmonary emphysema, 365, 374, 381.

Oesterlen, 23.

Ogston, 298.

Oil of turpentine in the treatment of catarrhal pneumonia, 230; inhalation of, in gangrene of lung, 430.

Ononis spinosa, decoction of, in the treatment of pulmonary emphysema, 405.

Oppolzer, 277, 285, 287, 317, 318, 389, 405, 430.

Origin of tubercle, 644.

Ott, 502.

Packing, cold, in the treatment of catarrhal pneumonia, 232.

Pagenstecher, A., 469.

Pain, in croupous pneumonia, 71, 168, in echinococcus of the lungs, 462; in pneumonia from embolism, 257; in pulmonary consumption, 528, 546, 552, 554, 585.

Palpation, in the physical examination of catarrhal pneumonia, 209; of croupous pneumonia, 75; of pulmonary consumption, 534; of pulmonary emphysema, 386.

Panum, 100, 245, 296.

Paracentesis of the pericardium, case of, 113.

Paracentesis thoracis in the diagnosis of pleuritic effusion complicating croupous pneumonia, 109.

Parasites of the Lungs, 462; Animal Parasites, 462; *the echinococcus*, 462; *symptomatology*, 462; influence of the size of the cysts, 462; the cough, 462; pain, 462; the dyspnœa, 462; emaciation, 463; distention of the thorax, 463; percussion sounds, 463; the vocal fremitus, 463; the respiratory sounds, 463; explanation of the symptoms, 464; *diagnosis*, 464; from pleuritic effusion, 464; from phthisis, 465; from new-growths, 466; *course of the disease*, 466; *prognosis*, 467; *the strongylus longevaginatus*, 467; its general characteristics, 467; *the cysticercus cellulosæ*, 468; Vegetable Parasites, 468; *the aspergillus*, 468; *sarcinæ*, 469; *oidium albicans and leptothrix pulmonalis*, 470.

Parent-Duchatelet, 20.

Parotid gland, inflammation of, a complication of croupous pneumonia, 122.

Pasteur, 418.

Pathogenesis of hypostatic processes in the lungs, 236; of pneumonia from embolism, 245.

Pathology of acute miliary tuberculosis, 620; of anæmia of the lungs, 264; of atelectasis, 326, of atrophy of the lungs, 338; of catarrhal pneumonia, 100; of croupous pneumonia, 32; of gangrene of the lung, 412; of hemorrhages of the lungs, 298; of hyperæmia and œdema of the lungs, 270; of hypostatic processes in the lungs, 240; of new-growths in the lungs, 433; of new-growths in the mediastinum, 446; of pneumonia from embolism, 252; of pulmonary consumption, 508; of pulmonary emphysema, 366.

Payet, 291.

Penzoldt, 251, 254, 255, 256, 259, 306, 312, 320.

Percussion in acute miliary tuberculosis, 623; in atelectasis, 332; in atrophy of the lungs, 341; in catarrhal pneumonia, 209; in croupous pneumonia, 78; after the third day in croupous pneumonia, 36; in echinococcus of the lungs, 463; in gangrene of the lung, 420; in the physical examination in hemorrhages of the lungs, 304; in hyperæmia of the lungs, 277; in hypostatic processes in the lungs, 242; in new-growths in the

lungs, 438, 441; in new-growths in the mediastinum, 452; in pulmonary consumption, 534, 584, 586, 587; in pulmonary emphysema, 364, 387.

Pericardium, diseases of, complicating catarrhal pneumonia, 215; complicating croupous pneumonia, 43, 112; disease of the pericardium in new-growths in the mediastinum, 455.

Pericarditis a complication of pulmonary consumption, 576.

Peritoneum, affections of, in croupous pneumonia, 43.

Peritonitis, a complication of pulmonary consumption, 576, 578.

Perl, 309.

Perls, 436.

Permanganate of potash, inhalation of, in gangrene of the lung, 430.

Perspiration in acute miliary tuberculosis, 620, 624; in pulmonary consumption, 562, 569.

Pertussis in the etiology of pulmonary consumption, 504.

Petechiæ in catarrhal pneumonia, 211.

Peter, 319.

Pfeilsticker, 221.

Pharynx, affections of, in pulmonary consumption, 546.

Phosphoric acid in the treatment of hyperæmia and œdema of the lungs, 284.

Phthisis, its diagnosis from echinococcus of the lungs, 465; its diagnosis from new-growths in the lungs, 442; a result of croupous pneumonia, 49, 130.

Physical examination in acute miliary tuberculosis, 623; in atelectasis, 332; in catarrhal pneumonia, 208; in croupous pneumonia, 35, 74; in gangrene of the lung, 420; in hemorrhages of the lungs, 304; in hyperæmia of the lungs, 277; in hypostatic processes in the lungs, 242; in parasites of the lungs, 463; in pneumonia from embolism, 257; in pulmonary emphysema, 383.

Pigment in atrophy of the lungs, 339.

Piorry, 63, 236, 237, 238, 241, 242, 536.

Pityriasis versicolor in pulmonary consumption, 562.

Pless, 445.

Pleura, affections of, in catarrhal pneumonia, 197; pleurisy, a complication of catarrhal pneumonia, 213; affections of the pleura in croupous pneumonia, 42, 87, 108; pleurisy with effusion, its diagnosis from echinococcus of the lungs, 464; affections of the pleura in gangrene of the lung, 414, 422; effusions into the cavity of the, in hypostatic processes in the lungs, 240; pleurisy, its differential diagnosis from new-growths in the lungs, 442; disease of the pleura in new-growths in the mediastinum, 455; affections of the, in pneumonia from embolism, 252, 253; pleurisy in the etiology of pulmonary consumption, 500; in the prognosis of pulmonary consumption, 598; a complication of pulmonary consumption, 574; pleurisy in tuberculosis, 658.

Pneumogastric nerves, pneumonia following section of the, 198.

Pneumonia, Catarrhal, 183; synonyms, 183; *bibliography*, 183; *history*, 183; *etiology*, 186; its alliance with the etiology of catarrhal bronchitis, 187; its frequency after diseases associated with catarrh, 187; the predisposing chronic diseases, 188; predisposing local diseases, 188; influence of age, 188; influence of sex, 189; of constitution, 189; of external influences, 189; of seasons, 190; influence of derangements in the walls of the thorax, 190; influence of catching cold, 190; *pathology*, 190; *symptomatology, course of the disease in general*, 190; the two forms of the disease, 190; origin of the acute form, 190; its symptoms, 191; the chronic form, 192; its symptoms, 192; the disease in adults, 193; a complication of diphtheria, 193; influence of previous state of health, 193; the so-called passive infiltrations, 194; the traumatic forms, 194; *anatomical changes*, 194; the bronchial catarrh, 194; changes in the lungs before the condition of true inflammation, 195; collapse of the lung, 195; its extent, 195; mode of progress, 196; emphysema as a complication, 196; changes in the lungs after the condition of true inflammation, 196; infiltrations, 196; the absence of granulation, 196; condition of the pus exuded, 196; condition of the seat of the infiltration, 196; formation of cavities, 197; affections of the pleuræ, 197; the bronchial glands, 197; recapitulation of the anatomical changes, 197; acute miliary tuberculosis as a complication, 197; the form of pneumonia after section of the pneumogastric nerves, 198; its description, 198; views as to its pathology, 198; its mode of regression, 199; *analysis of the symptoms*, 200; the temperature, 200; want of regular type of fever, 200; heights of temperature, 200; state of the circulation, 201; quality of the pulse, 202; changes in the heart, 202; the relations of tissue metamorphosis, 203; the appetite, 203; diarrhœa, 203; the spleen and liver, 203; collapse of the lung, 204; its mode of occurrence, 204; collapse in isolated portions, 205; the question of collapse as being essential to the development of catarrhal pneumonia, 206; emphysema, 208; results of physical examination, 208; inspection, 208; palpation, 209; fremitus, 209; percussion, 209; auscultation, 210; the exanthemata, 211; albuminuria, 211; noma vulvæ, 211; brain symptoms, 211; *complications and sequelæ*, 212; bronchitis, 212; pleurisy, 213; stenosis of the larynx, 213; tuberculosis, 215; croupous pneumo-

nia, 215; pneumothorax, 215; diseases of the pericardium and endocardium, 215; the chronic pneumonias as sequelæ, 216; bronchiectasia and emphysema, 216; pleuritic adhesions, 216; tuberculosis and rachitis, 216; intestinal disease, 216; later vulnerability of the lungs, 216; the heart, 216; glandular affections, 216; diagnosis, 216; the existence of catarrh, 216; consolidation of the lung, 217; the vocal fremitus, 217; inspection of the chest, 217; aggravation of the general symptoms, 217; the non-typical character of the fever, 217; the indefinite limitation of the disease, 217; differential diagnosis between collapse and catarrhal pneumonia, 218; between catarrhal and croupous pneumonia, 219; acute miliary tuberculosis, 219; *duration*, 220; time of death, 220; prognosis, 221; the rate of mortality, 221; influence of age, 222; extent of the pneumonic process, 222; powers of resistance of the patient, 222; other influences, 223; *treatment*, 223; prophylactic measures, 227; against catarrhs, 227; the use of baths 227; the dress, 228; the prevention of collapse, 228; fresh air, 228; in the acute stage, 228; in the chronic forms of the disease, 228; moisture of the air, 229; the use of muriate of ammonia, 230; of carbonate of soda, 230; of oil of turpentine, 230; the use of baths, 231; affusion over region of the medulla oblongata to produce increased respiratory efforts, 231; the use of cold packs, 232; treatment of the diminution of the cough, 232; emetics, 233; the use of apomorphine, 233; tartar emetic, 233; quinine and stimulants, 233; nutrition, 234; former methods of treatment, 234; treatment of the laryngitis, 234; residence in the country after recovery, 235.

Pneumonia, Croupous, 3; *bibliography*, 3; *history*, 4; *etiology*, 12; its proportion to other diseases, 12; influence of climate, 12; geographic distribution, 13; the season of the year, 13; influence of an insular or continental climate, 14; influence of elevation, 16; barometric pressure, 16; temperature, 17; moisture of the air, 17; influence of soil, 18; confinement in buildings, 18; influence of sex, 20; of age, 22; of constitution, 25; abortion following the disease, 25; influence of previous attacks, 26; of social position, 26; the disease among the lower animals, 27; question of epidemic occurrence of the disease, 27; the annual recurrence of the disease, 28; comparison of other diseases in point of time, 28; exciting causes, 30; traumatic causes, 20; chilling of the body, 31; inhalation of dust, 31; *pathology*, 32; general course of the disease, 32; mode of invasion, 33; duration of the disease, 33; the prodromata, 34; physical signs in first stage, 35; temperature, in first stage, 35; pulse and respiration, 35; condition of the skin, 35; the cough, 35; the sputa, 36; the urine, 36; percussion and auscultation on and after the third day, 36; abatement of symptoms at end of the first week, 37; crisis, 37; lysis, 38; mode of death, 38; *outline of the morbid anatomy*, 40; *the first stage: inflammatory engorgement*, 41; *the second stage: hepatization*, 41; *the third stage: purulent infiltration*, 42; affections of the pleura, 42; of the pericardium, 43; of the peritoneum, 43; of the bronchial glands, 43; of the bronchi, 43; of the heart, 43; changes in the stage of inflammatory engorgement, 43; in that of red hepatization, 43; source of the exudation, 44; changes in the stage of purulent infiltration, 47; caseation of the exudation, 49; variations in the form of the pneumonia, 50; variations in the site of the disease, 50; in the disease in childhood, 51; *analysis of the individual symptoms*, 52; *chronology*, 52; use of the thermometer, 53; the chill, 53; reckoning the days of the disease, 53; fixing the time of defervescence, 53; critical days, 54; the fever, 55; *temperature*, 55; general type, 56; during the forming stage, 56; at the height of the disease, 57; influence of locality of the disease, 57; influence of age, 57; of sex, 57; deviations from regular curve, 57; the quality of the fever, 58; influence of treatment upon the temperature, 58; the stage of defervescence, 59; *symptoms of the circulation*, 61; the pulse, 61; dicrotism, 63; the heart, 63; collapse, 65; the initial chill, 66; sweating, 66; the respiration, 67; relative frequency of pulse and respiration, 67; type of the respiration, 69; position of the patient, 69; the cough, 69; the pain, 71; pain on the non-inflamed side, 72; the sense of oppression, 73; the relations of the pain to the cough, 73; *physical examination*, 74; *inspection*, 74; cyanosis, 74; acceleration of the breathing, 74; difference in motion of the sides of the chest, 74; *mensuration*, 75; *palpation*, 75; palpatory percussion, 75; the vocal fremitus, 76; *percussion*, 78; variations in the tone, 78; Williams's tracheal tone, 79; rules for percussion, 80; methods of percussion, 82; *auscultation*, 82; the crepitant râle, 82; its occurrence in other diseases, 83; its frequency in pneumonia, 84; the râle redux, 84; its duration, 85; signs before the pneumonia has reached the surface of the lung, 85; their causes, 85; blowing respiration and bronchophony, 85; intensity of the râles, 85; cases which seem to indicate infiltration on the hitherto sound side, 86; points in diagnosis, 87; signs of pleuritis occurring in the disease, 87; the expectoration, 88; its characteristics, 88; the mucine, 88; the red blood-corpuscles, 88; casts of the bronchi, 89; pus-corpuscles, 89; epithelial cells,

89; chemical analysis of the sputum, 89; presence of sugar, 89; of bile pigment, 89; time of first appearance of the expectoration, 90; its quantity, 90; its absence, 90; expectoration with actual hemorrhage, 90; effects produced by œdema of the lungs, 91; the ceasing of expectoration at approach of death, 91; condition of nutrition, 92; examination of nitrogenous changes in the body, 92; discharge of carbonic acid, 94; metamorphosis of the inorganic matters, 94; influence of social position and occupation upon renewing work, 94; *organs of digestion*, 94; anorexia, 94; vomiting, 95; its causes, 95; constipation, 96; diarrhœa, 96; the tongue, 96; the stomach and intestines, 96; the liver, 96; the spleen, 96; the kidneys, 97; the urine, 97; abnormal substances in the urine, 99; *condition of the skin, eruptions*, 100; herpes facialis, 100; its frequency, 100; its prognostic value, 100; furuncles, 101; gangrene of the skin, 101; *cerebral and nervous symptoms*, 101; their causes, 101; the fever, 101; its special character, 102; influence of the site of the pneumonia, 102; influence of age, 103; of alcoholism, 103; of anæmia and debilitating treatment, 104; symptoms in intellectual persons, 104; local affections of the brain, 105; *blood and hemorrhages*, 106; changes in the blood, 106; various hemorrhages, 106; *complications*, 106; influence of local differences, 107; influence of hospital conditions, 108; affections of the respiratory and circulatory organs, 108; pleurisy with effusion, 108; its influence upon the pneumonia, 109; its diagnosis, 109; by paracentesis, 109; influence of pleurisy upon the mortality, 110; catarrh of the smaller bronchi, 110; emphysema, 111; heart complications, 111; valvular affections, 112; acute heart diseases, 112; chronic heart diseases, 112; pericarditis, 112; its mode of origin, 113; tapping the pericardium, 113; endocarditis, 114; valvular insufficiency and degenerations of the substance of the heart, 114; meningitis, 114; infectious meningitis, 115; its result, 116; icterus, 116; its importance in prognosis, 116; its anatomical cause, 117; dangerous symptoms due to icterus, 117; the epidemic "bilious pneumonia," 118; pneumonia a complication of kidney diseases, 119; rheumarthritis acuta, 119; intermittent fever, 119; pneumonia among drunkards, 119; its varieties, 119; pneumonia complicated by malaria, 121; its two forms, 121; inflammation of the parotid gland, 122; *different forms of pneumonia*, 123; abortive forms of pneumonia, 123; typhoid pneumonia, 124; its characteristics, 124; the cerebral form of pneumonia, 126; *relapses of pneumonia*, 126; *terminations*, 126; abscess of the lung, 126; its etiology, 126; its site, 127; diagnosis, 127; characteristics of the expectoration, 127; the results of abscess, 128; gangrene of the lungs, 128; its etiology, 128; general characteristics, 130; its results, 130; phthisis and cirrhosis, 130; etiology, 130; *mortality in croupous pneumonia*, 131; difficulty of arriving at just conclusions, 131; various statistics, 132; general estimate, 134; *diagnosis*, 134, its difficulties, 135; differential diagnosis, 136; *prognosis*, 138; influence of age, 138; from childhood to puberty, 138; the age of vigor, 139; the age of decline, 140; influence of sex, 140; of pregnancy, 140; influence of complications, 140; heart disease, 140; severity of the epidemic, 141; resisting power of the patient, 142; extent of the disease, 142; the fever, 142; pulse and respiration, 142; the cough, 142; the bronchial catarrh, 142; infiltration of the lung, 142; the expectoration, 143; eruption of herpes, 143; cerebral symptoms, 143; previous health, 143; *treatment*, 143; definition of the disease, 144; its etiology, in comparison with other diseases, 144; the relation between local and febrile symptoms, and the dependence of one upon the other, 146; the typical course of the disease, 147; indications for treatment, 150; the abortive method, 150; venesection, 150; causes of death, 152; insufficiency of heart action, 153; increased resistance in pulmonary circulation, produced by the exudation, 153; changes produced in and near the lung diminish the amount of force to be furnished by the heart, 154; diminution of surface of lung for exchange of gases, 155; the local disturbances brought to expression by the fever, 156; the fever inducing increased heart action, 156; elevation of temperature, 156; the pulse, 156; increased formation of carbonic acid gas, 156; degeneration of the muscular tissue of the heart, 156; prophylaxis against exhaustion of the heart, 157; control of already existing exhaustion, 157; treatment of the fever, 157; relative values of pulse and temperature as guides to treatment, 157; modes of combating the fever, 158; the question of direct abstraction of heat by the cold bath, 158; the so-called danger of catching cold, 159; the amount of heat to be withdrawn, 161; temperature of the bath, 161; its duration, 162; its application among the aged or among fat people, 162; among children, 162; stimulants before and after the bath, 163; indications for treatment by warm bathing, 164; the use of quinine, 164; tartar emetic and veratrine, 164; digitalis, 164; venesection, 165; reduction of heat by quinine, 165; mode of administration, 165; question of diet, 167; stimulants, 167; pain

and sleeplessness, 168; their treatment by hypodermic injection of morphine, 168; by quinine, 168; by chloral-hydrate, 169; the question of darkened rooms, 169; treatment of existing exhaustion of the heart, 169; of collapse, 170; manifestation of œdema, 171; œdema of the brain, 171; treatment of œdema, 173; venesection, 173; stimulants, 175; the use of camphor, 175; of musk, 176; the question of baths during cardiac exhaustion, 176; the use of quinine, 177; occurrence of collapse at the time of crisis, 177; its treatment, 177; preparations of iron, 177; *mortality*, 178; *complications and sequelæ*, 181; their treatment, 181; cirrhosis and gangrene of the lung, 182; administration of the oil of turpentine, 182.

Pneumonia from Embolism, 245; *bibliography*, 245; *history*, 245; *pathogenesis and etiology*, 245; source and nature of the emboli, 246; site of the embolus, 246; the heart as point of origin, 246; original formation in the veins of the general circulation, 247; the size as influencing the extent of the pneumonia, 248; influence of non-infectious plugs, 248; of infectious plugs, 248; method of development of hemorrhagic infarctions, 249; their location, 251; their shape, 251; the shape of abscesses, 251; *pathological anatomy*, 252; condition of the lung, 252; the site of the embolus, 252; the bronchi, 252; the pleura, 252; microscopic examination, 252; character of embolic abscess, 253; changes undergone by an infarction of long standing, 253; pulmonary gangrene, 253; the impossibility of suppuration of the infarction, 253; perforation into the bronchi, pleural cavity, or externally, 253; the greater liability of the right lung to infarctions, 254; *symptomatology and diagnosis*, 254; diseases which may give rise to the formation of thrombi, 255; the general symptoms, 255; increased frequency of respiration, 255; the fever, 256; chills, 256; the local symptoms, 255; the quality of the sputa, 256; length of continuance, 257; its amount, 257; the cough, 257; the pain, 257; physical exploration, 257; *prognosis*, 258; *treatment*, 259; the effect of the pleural exudation, 259; cardiac stimulants, 259; digitalis, 259.

Pneumonia, chronic, a complication of pleuro-pneumonia, 110; croupous, a complication of catarrhal pneumonia 215; form of, following section of the pneumogastric nerves, 198.

Pneumonia, lobular, see Catarrhal Pneumonia; a complication of kidney diseases, 119.

Pneumonia, its differential diagnosis from new-growths in the lungs, 442; in the etiology of pulmonary consumption, 499; a complication of pulmonary consumption, 574.

Pneumonia, cheesy lobar, in chronic tuberculosis, 673; desquamative, in tuberculosis, 664.

Pneumometry, 399.

Pneumothorax a complication of catarrhal pneumonia, 215; in gangrene of the lung, 422; its diagnosis from pulmonary emphysema, 394; in pulmonary consumption, 576, 598, 612; in pulmonary emphysema, 368; in tuberculosis, 682.

Pöhn, 447, 460.

Portal, 478, 522.

Position of the patient in croupous pneumonia, 69; of the body, prolonged maintenance of, a cause of hypostatic processes in the lung, 237.

Potassium, iodide of, in acute miliary tuberculosis, 630; permanganate of, inhalation of, in gangrene of the lung, 430.

Pravaz, 399.

Pregnancy, influence of, in prognosis of croupous pneumonia, 140.

Processes, healing, in tuberculosis, 682.

Prodromata of croupous pneumonia, 34.

Prognosis of atelectasis, 333; of catarrhal pneumonia, 221; of croupous pneumonia, 138; of echinococcus of the lungs, 467; of gangrene of the lung, 424; of hemorrhages of the lungs, 311; of hyperæmia and œdema of the lungs, 282; of hypostatic processes in the lungs, 243; of new-growths in the lungs, 442; of new-growths in the mediastinum, 456; of pneumonia from embolism, 258; of pulmonary consumption, 595; of pulmonary emphysema, 392.

Prophylaxis in catarrhal pneumonia, 227; against exhaustion of the heart in croupous pneumonia, 157; of gangrene of the lung, 428; of hemorrhages of the lungs, 317; of hyperæmia and œdema of the lungs, 284; of pulmonary emphysema, 396.

Prostate gland, caseous affections of, in the etiology of acute miliary tuberculosis, 617.

Puerperal condition in the etiology of pulmonary consumption, 505.

Pulmonary consumption, see Consumption.

Pulmonary emphysema, see Emphysema.

Pulse in acute miliary tuberculosis, 619, 623; in catarrhal pneumonia, 201; in croupous pneumonia, 35, 61; in prognosis of croupous pneumonia, 142; pulse and temperature as guides to treatment of croupous pneumonia, 157; pulse in gangrene of the lung, 420; in new-growths in the lungs, 439, 441; in new-growths in the mediastinum, 451, 455; in pulmonary consumption, 548, 599; in pulmonary emphysema, 364, 382.

Pus in catarrhal pneumonia, 196; croupous pneumonia, 89.

Quain, 440.

Quinine in the treatment of catarrhal pneumonia,

233; of croupous pneumonia, 164, 165, 168, 177; its influence upon the temperature in croupous pneumonia, 58; in the treatment of gangrene of the lung, 431; of œdema of the lungs, 287; of pulmonary consumption, 610; of pulmonary emphysema, 406.

Rachitis, a sequel of catarrhal pneumonia, 216.
Rainey, 372.
Râles in acute miliary tuberculosis, 619; crepitant râles in croupous pneumonia, 82; the râle redux in croupous pneumonia, 84; râles in hyperæmia of the lungs, 277; in hypostatic processes in the lungs, 242; in pulmonary consumption, 585, 586, 587.
Ramadge, 375.
Rasmussen, 304.
Rayer, 468.
Reichenhall, mineral waters of, in the after-treatment of atelectasis, 336; mineral waters of, in pulmonary consumption, 604.
Reid, 478.
Reinay, 271.
Reinerz, mineral waters of, in pulmonary consumption, 606.
Reinhardt, 480, 561, 660.
Relapses of croupous pneumonia, 126.
Respiration in acute miliary tuberculosis, 622; frequency of, in first stage of croupous pneumonia, 35; in later stages of croupous pneumonia, 67, 74; in prognosis of croupous pneumonia, 142; in echinococcus of the lungs, 463; in new-growths in the lungs, 439, 440; in new-growths in the mediastinum, 450; in pneumonia from embolism, 255; in pulmonary emphysema, 376.
Remak, 89, 468.
Rhazes, 8, 477.
Rheumarthritis acuta, a complication of croupous pneumonia, 119.
Ribs, changes in the cartilages of, in the etiology of pulmonary consumption, 485.
Richter, A. G., 11, 185.
Riegel, 395, 399, 404, 409, 445, 448, 459.
Riesell, 90, 92.
Rilliet, 186, 413, 500, 504, 549.
Rindfleisch, 43, 44, 47, 48, 49, 193, 239, 297, 339, 370, 371, 409, 436, 437, 487, 574.
Rindfleisch on chronic and acute tuberculosis, 633.
Robin, 275, 276, 469.
Roccas, 186.
Roger, 83, 406.
Rogers, 318.
Rokitansky, 11, 41, 240, 246, 253, 264, 271, 274, 275, 292, 302, 327, 328, 343, 345, 346, 350, 355, 356, 375, 415, 434, 480, 500, 620, 633, 636, 656.
Rosenstein, 279.
Rosignol, 370.
Rossbach, 454.
Ruehle on acute miliary tuberculosis, 614; on pulmonary consumption, 473.
Runge, 607.
Rush, 26.

Salt in the treatment of gangrene of the lung, 429; of hemorrhages of the lung, 319.
Sarcone, 10.
Schauer, 469.
Schmidt, 435.
Schnyder, 436.
von Schrötter, L., 60.
Schultze, F. E., 649.
Schüppel, 44, 207, 212, 574, 641.
Scoda, 11, 182, 210, 274, 319, 343, 428, 429, 446, 454, 537.
Scrofula in the etiology of pulmonary consumption, 484; its relations to tuberculosis, 635.
Seasons, influence of, in etiology of catarrhal pneumonia, 190; influence of the, upon croupous pneumonia, 14.
Seitz, 77, 268, 293, 542.
Senator, 92.
Senega in the treatment of catarrhal pneumonia, 232; of hyperæmia and œdema of the lungs, 286.
Servius, Petrus, 8.
Sequelæ of catarrhal pneumonia, 212; of croupous pneumonia, 181; of hemorrhages of the lungs, 307.
Sex, influence of, in catarrhal pneumonia, 189; in croupous pneumonia, 20, 140; in emphysema of the lungs, 348; in gangrene of the lung, 408; in hemorrhages of the lungs, 294; in new-growths in the mediastinum, 445; in pulmonary apoplexy, 298; in pulmonary consumption, 506, 526.
Sickliness in the etiology of pulmonary consumption, 484.
Skin, affections of, in acute miliary tuberculosis, 623; in catarrhal pneumonia, 211; in croupous pneumonia, 35, 100; derivation to the skin in œdema of the lungs, 286; affections of the skin in pulmonary consumption, 561.
Skrzeczka, 436.
Slawjansky, 470.
Sluyter, 469.
Soda, carbonate of, in treatment of catarrhal pneumonia, 230; in pulmonary consumption, 605.
Soden, mineral waters of, in pulmonary consumption, 604.
Soil, condition of, in the etiology of pulmonary consumption, 490.
Sommerbrodt, 309, 403.
Spinal cord, affections of, in pulmonary consumption, 561.

Spleen, affections of, in acute miliary tuberculosis, 619, 621, 625; in catarrhal pneumonia, 203; in croupous pneumonia, 96, 124; in pulmonary consumption, 559, 581, 600; in pulmonary emphysema, 374.
Splenization of the lungs in atelectasis, 328; in hyperæmia, 271.
Sprengel, Kurt, 10.
Squill in the treatment of pulmonary emphysema, 405.
Stark, 478.
Steffen, 186, 189, 215, 221, 328.
Steiner, 222.
Stenosis of the larynx, a complication of catarrhal pneumonia, 213.
Stethography, 399.
Steudener, 45.
Stimulants, cardiac, in the treatment of acute miliary tuberculosis, 630; in the treatment of catarrhal pneumonia, 233; in connection with cold baths in croupous pneumonia, 163; in connection with diet in croupous pneumonia, 167; in connection with exhaustion of the heart in croupous pneumonia, 175; in the treatment of pneumonia from embolism, 259; of pulmonary emphysema, 406.
Stokes, Wm., 125, 186, 264, 396, 414, 429, 437.
Stoll, 10, 118.
Stomach, affections of, in croupous pneumonia, 96; in gangrene of the lung, 420; in pulmonary consumption, 551, 579; in pulmonary emphysema, 374.
Stromeyer, 76.
Strychnine in the treatment of pulmonary emphysema, 396.
Subbotin, 223.
Sugar in the expectoration of croupous pneumonia, 89.
Sulphuric acid in the treatment of hyperæmia and œdema of the lungs, 284.
Sweating in croupous pneumonia, 66.
Sweating at night in pulmonary consumption, 586.
von Swieten, 8, 185, 307.
Sydenham, 9, 183, 185, 307.
Sylvius, 307, 477.
Symptomatology of acute miliary tuberculosis, 622; of anæmia of the lungs, 264; of atelectasis, 330; of atrophy of the lungs, 340; of catarrhal pneumonia, 190; of croupous pneumonia, 52; of echinococcus of the lungs, 462; of gangrene of the lung, 415; of hemorrhages of the lungs, 302; of hyperæmia and œdema of the lungs, 276; of hypostatic processes in the lungs, 241; of new-growths in the lungs, 437; of new-growths in the mediastinum, 450; of pneumonia from embolism, 254; of pulmonary consumption, 517; of pulmonary emphysema, 376.

Tabarie, 399.
Tannin in the treatment of hemorrhages of the lungs, 318.
Tartar emetic in the treatment of catarrhal pneumonia, 233; of croupous pneumonia, 164; of hyperæmia and œdema of the lungs, 285.
Teeth, affections of, in pulmonary consumption, 564.
Temperature in acute miliary tuberculosis, 619, 624; in catarrhal pneumonia, 192, 193, 200; of the air in croupous pneumonia, 17; of the body in croupous pneumonia, 35, 53, 55; in its prognosis, 142; temperature and pulse as guides to treatment of croupous pneumonia, 157; temperature in gangrene of the lung, 420; in hemorrhages of the lungs, 306; in hypostatic processes in the lungs, 242; in pneumonia from embolism, 256; of the air, in the etiology of pulmonary consumption, 491; of the body in pulmonary consumption, 567; of the body in the prognosis of pulmonary consumption, 599.
Testicles, caseous affections of, in the etiology of acute miliary tuberculosis, 617.
Thierfelder, 172.
Thirst in pulmonary consumption, 553.
Thomas, 54, 56, 57, 79, 100, 101.
Thorax, compression of, in pulmonary emphysema, 404.
Throat, affections of, in pulmonary consumption, 553.
Thrombosis a cause of hyperæmia of the lungs, 268.
Thyroid gland, affection of, in new-growths in the mediastinum, 455.
Tissot, 10.
Tone, tracheal, of Williams, 79.
Tongue, affections of, in croupous pneumonia, 96; in pulmonary consumption, 552.
Tophoff, 54.
Tracheal tone of Williams, 79.
Trallianus, Alexander, 7.
Traube, 54, 69, 87, 116, 117, 127, 128, 129, 198, 287, 349, 410, 416, 417, 419, 426, 427, 428, 508, 520, 547, 560, 622.
Treatment of acute miliary tuberculosis, 629; of anæmia of the lungs, 265; of atelectasis, 335; of atrophy of the lungs, 342; of catarrhal pneumonia, 223; of croupous pneumonia, 143; of gangrene of the lung, 428; of hemorrhages of the lungs, 316; of hyperæmia and œdema of the lungs, 284; of hypostatic processes in the lungs, 243; of new-growths in the lungs, 443; of new-growths in the mediastinum, 460; of pneumonia from embolism, 259; of pulmonary consumption, 600; of pulmonary emphysema, 396.
Trousseau, 319, 429.
Tubercles, their anatomy, 642; their definition, 643; their origin, 644; the tubercle granulum, 648.
Tubercles, miliary, as secondary formations in pulmonary consumption, 572.

Tuberculosis: Chronic Tuberculosis, 633; *tuberculosis and scrofula*, 633; *history*, 633; *scrofula*, 635; its characteristics, 635, *how scrofulous persons become tuberculous*, 639; *disease of the lymphatic glands*, 640; *the anatomy of tubercles*, 642; *definition of tubercle*, 643; *origin of tubercles*, 644; conclusion, 646.

Chronic Tuberculosis. Phthisis, 648; *commencement of the disease*, 648; *catarrhal bronchitis and tuberculosis*, 648; *the tubercle granulum*, 648; its characteristics, 648; its situation, 649; definition of an acinus of the lung, 649; mode of formation of the tubercle granulation, 650; the softened centre of the granule, 652; erroneous explanations of the granule, 653; *catarrh of the bronchi*, 653; its general characteristics, 654; the cause of the disease in apices of the lungs, 654; influence of their position, 655; *hemorrhages and pleurisy*, 656; the initial hæmoptysis, 656; its causes, 656; character of the blood, 658; results, 658; the pleurisy, 658; its characteristics, 658; development of new blood-vessels, 659; *the later stages of the disease: cheesy broncho-pneumonia*, 660; the crude tubercle of authors, 660; the lesions of the mucous membrane, 661; tubercular ulceration of the mucous membrane, 662; the peribronchial lesions, 663; desquamative pneumonia, 664; the pulmonary epithelium, 665; cheesy degeneration, 668; *principal varieties of pulmonary phthisis*, 668; the ordinary chronic variety, 668; the acute varieties, 670; atelectasis, 670; œdema, 671; slate-colored induration, 672; cheesy lobular masses, 672; cheesy lobar pneumonia, 673; *cavities*, 676; definition, 676; bronchiectatic conditions, 676; the early stage of the formation of cavities, 677; the opening and enlargement of cavities by the mechanism of respiration, 678; enlargement of cavities, 679; the fully formed cavities, 681; hæmoptyses, 681; pneumothorax, 682; *healing processes*, 682; calcification, 682; shrinkage of the infiltration, 683; *the rarer varieties*, 687; the disease in children, 687; affections of the lymphatics, 688; cirrhosis of the lungs, 689; bronchophthisis, 690.

Acute Tuberculosis, 691.

Tuberculosis, Miliary, Acute, 614; *introduction*, 614; *etiology*, 616; absorption of caseous matter, 616; location of the disease, 617; *general description and course of the disease*, 618; *pathological anatomy*, 620; *analysis of individual symptoms*, 622; the cough, 622; the expectoration, 622; character of the breathing, 622; dyspnœa, 622; physical signs, 623; percussion, 623; auscultation, 623; the pulse, 623; the skin, 623; fever, 624; perspirations, 624; the nervous symptoms, 624; symptoms of the digestive organs, 625; the spleen, 625; the appetite, 625; the discharges from the bowels, 625; the urine, 625; *complications*, 625; *diagnosis*, 626; differential diagnosis from typhoid fever, 626; from acute bronchial catarrh, 628; *duration, terminations, prognosis*, 629; *treatment*, 630; iodide of potassium, 630; cardiac stimulants, 630.

Tuberculosis as a cause of hyperæmia of the lungs, 269; a complication of catarrhal pneumonia, 215, 216; miliary, acute, a complication of catarrhal pneumonia, 197.

Tulpius, 293.

Tumors as causes of hyperæmia of the lungs, 268.

Turpentine, oil of, in treatment of catarrhal pneumonia, 230; in treatment of cirrhosis of the lung in croupous pneumonia, 182; in gangrene of the lung in croupous pneumonia, 182; inhalation of, in gangrene of the lung, 430; in treatment of hemorrhages of the lungs, 319.

Typhoid form of pneumonia, 124.

Uhle, 372.

Urine in acute miliary tuberculosis, 620, 625; in croupous pneumonia, 36, 97; in pulmonary consumption, 559; in pulmonary emphysema, 364, 382.

Uva ursi in pulmonary emphysema, 405.

Vaccination in the etiology of scrofula as a cause of pulmonary consumption, 485.

Valvular changes in the heart in croupous pneumonia, 114.

Veith, J. E., 27.

Venesection in abortive treatment of croupous pneumonia, 150; its antifebrile action in croupous pneumonia, 165; in treatment of œdema of the lung in croupous pneumonia, 173; in hemorrhages of the lungs, 316, 320; in hyperæmia and œdema of the lungs, 285, 286, 287; in pulmonary emphysema, 405.

Veratrine in treatment of croupous pneumonia, 164.

Vibriones in gangrene of the lung, 418.

Villemin, 372, 495, 634.

Virchow, 45, 46, 245, 246, 274, 275, 292, 295, 297, 306, 326, 343, 417, 434, 447, 448, 456, 468, 469, 470, 480, 481, 521, 634, 636, 637, 640, 642, 655, 660, 673.

Vivenot, 399.

Vocal fremitus in the physical examination of croupous pneumonia, 76; of echinococcus of the lungs, 463; of hypostatic processes in the lungs, 242; of new-growths in the lungs, 438, 441; of new-growths in the mediastinum, 452, 456.

Vogel, J., 97, 98.

Vogel, S. G., 11.

Voit, 93.

Volkmann, 45, 410.

Vomiting in croupous pneumonia, 95; in gangrene of the lung, 420; in pulmonary consumption, 552.
Vulva, noma of in catarrhal pneumonia, 211.

Wagner, 46, 194, 372, 434.
Waldenburg, 310, 346, 400, 404, 477, 543, 617, 635.
Waldenburg's apparatus for the administration of condensed air, 400.
Waller, 616, 618, 625, 626, 630.
Walshe, 83, 89, 351, 383, 436.
Wartena, 465.
Water, cold, baths of, in treatment of croupous pneumonia, 158; warm, baths of, in treatment of croupous pneumonia, 164.
Waters, chalybeate, in the prophylaxis of hemorrhages of the lungs, 317; mineral, in prophylaxis of pulmonary consumption, 604.
Waters, A. T. H., 318.
Weber, H., 319.
Weight, loss of, in the prognosis of pulmonary consumption, 599.
Weinburger, 414.
Welker, 469.
Williams, 79, 83, 353.
Williams's tracheal tone, 79.
Wintrich, 72, 75, 76, 78, 82, 83, 84.
Woillez, 385, 534.
Wolff, 319.
Wunderlich, 54, 57, 58, 59, 64, 67, 106, 123, 125, 292, 370, 372, 449, 452, 630.

Zenker, 52, 156, 275, 283.
Von Ziemssen, H., 25, 31, 35, 50, 51, 54, 55, 56, 57, 61, 75, 82, 86, 138, 186, 189, 190, 195, 198, 208, 218, 220, 221, 222, 234, 235, 355, 384, 500.
von Ziemssen, W., 12, 13, 15, 16, 21, 22, 28.

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